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INDUSTRIAL DERMATOSES*

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Industrial dermatology has provided the material for innumerable papers in recent years. American dermatologists, in particular, seem to be in open competition to reach the pages of medical journals with descriptions of new chemicals, new sensitisers, new industrial processes, and new patterns of occupational dermatitis. All this is very useful, inasmuch as it draws attention early to new industrial hazards. To the factory medical officer and the industrial dermatologist the determination of the specific irritant which is responsible for the incidence of dermatitis in a particular industry is immensely important, and very often by substitution of one solvent or one cutting-oil by another it is possible to reduce that incidence.

One criticism that may be offered is that all this cards indexing of industries and their respective dermatoses tends to create the impression that the various forms of dermatitis are type-specific and region-specific, which is not by any means the case. While it is possible to reduce a particular industrial hazard to a chemical or physical formula, the human material, which gives this problem its more inconstant factor, cannot be reduced to any formula. The same irritant might produce one type of eruption in workers of a younger age-group and a different type in older workers. The dermatitis pattern in any industry might vary, as regards areas affected and type of eruption, with the conditions of temperature and humidity, the kind of garment worn at work, the race, sex, and state of nutrition of the workers. In eczematous contact-type dermatitis all the vagaries of allergy become applicable. A workman might react when he is tired and be blissfully unaffected when he is rested; the psychological atmosphere in which he works might influence his sensitivity threshold; a female worker might show heightened sensitivity during her menstrual periods and at the menopause. When more than one etiological factor is involved, the original clear-cut pattern might become obscured, for example, an eczematous dermatitis due to contact with nickel might be altered beyond recognition by a secondary infective dermatitis.

In the final analysis the primary aspect of the problem is that in all industries, under all conditions of work, some workmen are liable to suffer from dermatitis. To the dermatologist-at-large the problem presents itself at an earlier stage as a differential diagnosis between industrial dermatitis in its wider sense and unrelated skin diseases to which the workman is subject in common with the general population.

THE HISTORY

The history of the case would be expected to have an important bearing on the diagnosis of industrial dermatitis. To the unitiated it might commend itself as a simple question and answer routine: Does the eruption pre-date or post-date the employment? What is the nature of the substance or substances to which the workman is exposed? What are the conditions of work? Is there anything in the workman's hobbies or domestic circumstances which might be relevant? Are there any other workmen in the same employment similarly afflicted?

In theory this is all very simple and obvious; in practice the investigator's path is often thickly strewn with 'red herrings'. At the best of times history-taking in dermatology is not the joyful interlude it is supposed to be. A dermatologist making his first contact with a patient is apt to find himself in a strange world of anachronisms with humours, vapours, blood impurity, and excessive acidity. The idea of contact sensitivity appears too remote and too revolutionary. Any inquiry into a possible relationship to gardening or the use of cosmetics is met with ill-concealed scepticism. The attitude is, why doesn't he tell me to stop eating tomatoes and give me something to clear my blood? It is not easy

* This paper was read at the Medical Congress of the Association held at Cape Town in September 1949.

to convince a patient that an innocuous substance to which he has been exposed for many years can suddenly become an allergen. One has tried quoting to patients illustrative cases, such as the one described by Hellerstrom[1] of contact sensitivity to mushrooms which only manifested itself after 20 years of innocuous contact. The reaction usually is that such things can only happen in Sweden.

By strange contrast the patient who is alleged to be suffering from Baker's Itch or Imbuya poisoning is pathetically eager to prove that his skin trouble is due to contact with something at his work. The merest hint that some preliminary investigation may be necessary before the diagnosis is accepted, brings the dermatologist's competence into question and induces a state of passive resistance. The workman has never had anything wrong with his skin before he adopted his present employment; his whole family for generations have been free from dermatological taint; gardening cannot possibly have anything to do with it, because he only does a little of it over the week-end; there is nothing wrong with his general health; his blood is pure because when he cuts himself it heals beautifully; half the workmen in his factory suffer from this complaint; anyway, a man who used to live in a town in England where there are many such industries told him that this is dermatitis. Incidentally, the statement that other workmen are similarly affected must not be accepted without verification, as the others might be afflicted with anything from leprosy to chilblains. To the lay public only two skin diseases are known. The unemployed suffer from eczema and those in trades suffer from dermatitis.

CLASSIFICATION

As distinct from the card-indexing already referred to, a useful approach to the clinical evaluation of cases is to provide oneself with a simple classification into several large etiological groups, providing always it is borne in mind that more than one etiological factor may be operative in a particular case. The following groups are suggested:

Group 1—*Effect of Chemical or Physical Agents.* Workmen may be affected in a variety of ways by chemical or physical agents, which inflict trauma on the exposed parts of the skin. As examples may be cited the corrosive and caustic action of acids and alkalies, the effects of extreme heat and cold, exposure to actinic rays and X-rays. Generally such agencies act by producing necrosis or atrophy of tissue. A notable exception is Miliaria Rubra which is undoubtedly an industrial disease in deep-level miners and stokers. The work of O'Brien[2] has shown that in this condition the pathology is that of follicular plugging and cystic dilatation of sweat ducts, quite unlike the atrophy and necrosis of this class.

A somewhat different form of trauma is met with in industries in which workmen are exposed to so-called 'primary irritants'. This term 'primary irritant', has never been clearly defined. It might be said that substances which will produce irritation in most people who are exposed to them, or which will give positive reactions on patch testing in almost 100% of test subjects, might be regarded as such, but neither of these criteria are exact. A chemical in a stated concentration might prove irritating to most workers who handle it for long periods, but might be innocuous in another process in which it is only handled casually and contact is limited to short periods.

Schwartz, Tulipan & Peck[3] can do no better than define a primary irritant as an agent which will cause dermatitis by direct action on the normal skin at the site of contact if it is permitted to act in sufficient intensity or quantity for a sufficient time.

If we apply the criterion of patch tests a measure of uncertainty is introduced by the time of application of the patch test and the actual percentage of positives that qualifies a substance as a primary irritant. For instance, Sullivan and King[4] have shown that 25% podophyllin resin in oil gives a positive patch test in 48 hours in 69% of test subjects. Is 69% to be regarded as a sufficiently high percentage for our purpose and, if not, would the position alter if the patch test were left on for 72 hours? Again, patch tests with soap solution will give a very high percentage of positives and yet one would hardly regard soap as a primary irritant on the undamaged skin.

Another perplexity that applies to this class of dermatosis is this: It is almost a social law that manual workers have rough, calloused and harsh hands, while an office worker has soft, smooth hands. At what stage does this rough hand of the working man become an industrial dermatosis? If we say that it does so only when the workman complains of discomfort, then we are leaving the whole assessment of the case to the nebulous criterion of subjective sensation.

Mumford[5] classifies primary irritants as (1) Fat solvents, (2) Keratin solvents, (3) Abrasives. Examples of fat solvents are alcohols, soaps, and carbon tetrachloride, as used by dry-cleaners. Keratin solvents include ammonia and caustic soda. Abrasives are used in polishing metals and other articles. Some of these abrasives, e.g. the trichloroethylene compound, are both abrasive and degreasing.

Group 11—*Eczematous Dermatitis or Contact Dermatitis* (these terms are here used synonymously). In this group a workman becomes sensitised at his work to a substance which is innocuous to the majority of workers. This form of allergic sensitivity may take many years to develop, and to begin with is specific for the substance to which the workman has become sensitised. The future of such a workman is unpredictable. It is well known in industry that many workmen become 'hardened', i.e. after a period of reaction they become desensitised by some unexplained process and can continue with their work. It is not uncommon for such workers to show some recurrence of their sensitivity after a period of idleness. Geoffrey Hodson[6] advocates that workers who have been away from work because of dermatitis should be encouraged to return to work early in order that this process of hardening be allowed to develop.

As it is not within the scope of this paper to deal with treatment, it may be mentioned at this stage that this process of hardening can sometimes be artificially imitated by subjecting the workman to a series of patch tests with the offending allergen. This has been done with imbuya workers who were taken off work for a

period and had patches of imbuya dust applied every few days until they ceased to show a positive reaction, Such workmen were then able to resume their trade.

In those workmen in whom 'hardening' does not take place, the sensitivity is liable to be progressively intensified by further exposure and may even cease to be specific. In other words, the workman may after a time react to substances of a similar chemical composition.

If such a workman ceases further exposure at this stage his skin usually returns to normal, but in some cases a phenomenon known as 'residual dermatitis' makes its appearance. In such cases, although contact with the offending allergen has stopped, the skin in the areas originally exposed remains eczematous and may even flare up after having almost completely subsided. There is no satisfactory explanation of this phenomenon. In some cases it may be that the original sensitisation has become group-specific and the patient is now reacting to substances related to the original allergen. In others he may have become intolerant of soap and water. It is also remotely possible that the primary contact allergen is still operating as an inhalant or a food allergen. However reasonable these explanations may be, they do not account for every case. If one were to accept the theory that an eczema is the outward expression of a chemical process which takes place in sensitised skin, one could visualise a situation in which the formation of histamine-like or other chemical substances becomes a permanent function of the eczematised skin. This would find an analogy in the behaviour of the thyroid gland which hypersecretes during periods of mental stress and may go on hypersecreting when the mental stress has been removed.

So far we have dealt with the type of eruption which is still limited to the area exposed to the irritant. If, however, the workman continues to be exposed to the noxa for a prolonged period, in spite of the increasing intensity of his sensitivity reaction, and more especially if he is subjected to injudicious therapy, he may develop a persistent irritable eczematous state, which spreads beyond the area of initial exposure, even to the point of becoming generalised. At this stage all specificity of allergen and reaction is lost and the patient may react to almost anything that comes in contact with his skin.

Again we find that this state has never been satisfactorily explained. If we take the hypothesis of a chemical substance a stage further, we could postulate that in this state such a chemical substance is overflowing into the general circulation and thus disseminating the eczematous reaction. The work of Haxthausen[7] on skin transplants in identical twins offers some corroboration of the theory of a circulating substance of unknown nature.

This generalised eczematous state is often referred to now as 'sensitisation dermatitis'. It is in many ways an unfortunate term. There is a growing tendency to use the term loosely as eczema and seborrhoeic dermatitis were used in the past. In any case it presupposes that there is also a form of contact-type eczematous dermatitis which is not associated with sensitisation. This is manifestly untrue, as every form of eczematous dermatitis must have had previously at least one innocuous exposure. Perhaps if this type were referred to as chronic eczematous dermatitis, as distinct from the acute type in which removal of the allergen results in a complete reversal to normal, it would be less likely to lead to confusion.

Patch tests have now become so much an integral part of the dermatologist's approach that it seems almost unnecessary to add that this useful test only applies to the diagnosis of the type of dermatitis in which a true and specific allergy is demonstrable. Text-books of dermatology devote ample space to the methods of applying patch tests and the possible misinterpretations that arise from their use, and one need only add that in this group the patch test, skilfully used, is the ultimate criterion on which the diagnosis is based. We can still find in text-books of dermatology lengthy and detailed descriptions of the clinical differentiation between such entities and pseudo-entities as eczema of the hands, cheiropompholyx, infectious eczematoid dermatitis, pustular dermatitis, dermatophytids, and contact dermatitis. All this postulates a degree of clinical exactitude which would only be possible if the points of differentiation were less inconstant and the area of overlap not so wide. Contact dermatitis may appear in the guise of any of these eruptions and can only be differentiated with certainty by patch tests.

Group III—Occupational Infections of the Skin. A large number of infections and infestations of the skin are contracted by workers in the course of their employment. A list of these includes anthrax, actinomycosis, tuberculosis verrucosa cutis, animal ringworms, orf, moniliasis, butcher's pemphigus, *erysipeloid* of Rosenbach, grass-mite dermatitis, cheese-mite dermatitis, etc., but the most important occupational skin infection is the post-traumatic infective dermatitis. In this type the trauma is so insignificant that *per se* it does not constitute an occupational dermatosis, but the cumulative effect of these repeated minute traumata—the scratches, the cuts, the abrasions—is to upset the equilibrium between the skin and the surface organism. Other important forms of trauma are maceration as a result of constant immersion of the hands in water and the fissuring effect produced by evaporation of liquids from the surface of the skin.

Percival[8] describes post-traumatic infective dermatitis as an eruption which commonly affects the hands and forearms and consists of one or more plaques of pustulovesicular eczema. The edge of these patches is in the form of a circle or segments of a circle, and the patches are sharply demarcated from the surrounding skin. The palms are more commonly invaded than in the eczematous type, because the pre-existing trauma reduces the protective efficiency of the thick stratum corneum and allows the infective process to spread under this stratum, which may eventually desquamate over the whole area affected.

It might be argued that this type of dermatitis does not differ fundamentally from any other dermatitis which had become complicated by a secondary bacterial infection, but whereas in the ordinary secondary impetigo the invading organisms spread themselves diffusely over an eczematous surface, in the post-traumatic infective dermatitis the eruption is circumscribed because the

dermatitis is primarily infective and the pre-existing trauma merely acts as portal of entry.

Another somewhat theoretical distinction is that when a bacterial infection becomes superimposed on an eczema reaction, the skin also shows an allergic sensitivity to the invading organisms. In post-traumatic infective dermatitis no such bacterial allergy exists. Whether these points of differentiation justify putting this dermatosis into a separate group is debatable, but it will generally be admitted that clinically it forms a very distinct entity.

Group IV—Miscellaneous. While most dermatoses of industrial origin fall into one or other of the first three groups, it will be found useful to have a fourth group of miscellaneous skin diseases which cannot easily be assigned to the other groups, either because they are etiologically indeterminate, or because they present a composite picture, or because the skin reaction has strikingly distinctive features.

As an example of the indeterminate type might be quoted the all too common case of the workman who presents himself with a chemical dermatitis attributable to the proprietary ointments which he had applied in an honest if somewhat misguided attempt to cure some skin disease, which is now claimed to have been originally of industrial origin. However well-founded that claim may be, the position now is that the dermatologist cannot apply the standards of examination and test to the original condition at this stage.

Another example is the type of case which presents itself as a nondescript eruption suggesting a chronic impetigo or some other non-industrial dermatosis, but which the workman attributes to heat or dust at his work. Moreover, he is often able to demonstrate that a rest from work produces a marked change for the better in his condition.

The composite type of eruption is well illustrated by two common dermatoses—Baker's Itch and Oil Dermatitis. In Baker's Itch the eruption may be partly due to an allergic sensitivity to flour, partly to the degreasing action of substances used in cleaning tins, and partly to the constant exposure to alkaline soaps. Similarly, oil acne is generally regarded as due to the mechanical blockage of the follicles, the irritative effect of oils inducing a hyperkeratosis at the mouths of the sebaceous ducts, and a secondary pyogenic infection. The picture becomes even more complex in cutting-oil dermatitis, or 'mystic dermatitis'. These cutting-oils contain an oil, water, and an emulsifier and the skin reaction is partly due to oil and partly to the degreasing action of the emulsifier when soap is used subsequently.

Among the distinctive effects of occupations on the skin, the pitch wart and the tar epithelioma are well known. In South Africa this type of occupational disease is of particular importance as a strikingly similar condition is produced by prolonged and excessive exposure to sunlight. If the question of recognising solar keratosis and epitheliomata as an industrial disease in bricklayers, plasterers and farm hands ever arises in South Africa, there will be no cogent reason for withholding such recognition. The whole subject of sun-sensitivity and sun damage might present us with some thorny problems in the future. Conditions like lupus erythematosus and colloid degeneration of the skin will be claimed as industrial dermatoses, with a large measure of justification, and the dermatologist will presumably be left with the onus of deciding to what extent occupation is responsible and how much of the damage can be blamed on the general effect of our climate.

A few dermatoses are placed in this miscellaneous group because of the unusual way in which the skin is affected at work. In glass wool dermatitis a micropapular eruption of the hands and wrists results from the penetration of the skin by fine particles of glass fibre and glass dust. A very similar condition of the fingers is produced by fine particles of asbestos.

Occupational leukoderma is another unusual affection of the skin produced by the use of heavy rubber gauntlets in tanneries. The depigmentation is caused by an anti-oxidant (Monobenzyl ether of Hydroquinone) which is added to the rubber.

Another pigmentary change of the skin is the melanosis which is encountered in workers exposed to tar and pitch. This dermatosis could also serve as an example of exposure to some chemical substance combined with photosensitisation. Incidentally, photosensitisation might account for some failures to demonstrate allergic contact sensitivity by patch tests. Evidence of this presented itself recently in a case of imbuya dermatitis in whom a positive patch test was only obtained after exposing the test area to ultra-violet light before applying the patch.

PRE-EMPLOYMENT SELECTION

Lately the term 'pre-employment selection' has made its appearance in the medical literature[11] together with the usual loose statements about dry skins and oily skins. As a final essay in classification we might perhaps relegate this concept to its proper place among the ideas that have a reasonable theoretical basis, but very little practical application. Obviously it is impossible by any test known to dermatology to predict what an individual will react to unless he had previously been exposed to and sensitised to that substance. Nor can one assess his capacity for 'hardening', or his psychological adaptability. Even assuming that this applies only to selection for occupations in which the hazard is traumatic, there still remains the palpable fallacy that a workman declared eligible by virtue of his skin texture, when he is young, will remain eligible when he is older and his skin has become more vulnerable.

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EDITORIAL

THE MEDICAL COUNCIL'S ANNUAL LEVY

From time to time Federal Council has considered the validity of the Medical Council's requirement that members of the profession pay an annual fee of £2 in order to keep their names on the medical register, in addition to the initial registration fee.

Indeed, the matter was raised at the most recent meeting of Federal Council, because many of our colleagues felt that the position was open to question and have found irksome the requirement to pay this annual fee.

In part the irritation probably derives from the manner in which the annual levy was introduced, there having been no previous consultation with or notice to the profession of the Council's intention to impose an annual payment.[1]

The legal validity of the Council's requirement has also at times been in question in certain quarters and it may well be that the technical position is not clear beyond all doubt.

We do know that in terms of sec. 95 (2) of the Medical, Dental and Pharmacy Act, the Minister has power to make refunds to the Council in terms of sec. 94 (2) (c). We do not know if the Minister has at any time made such refunds but, in any event, such payments would have covered only the very limited expenses of five medical members of the Council.

The annual levy was imposed presumably because the Council needed to increase its funds.[1, 2] The introduction of an annual payment was accompanied by a reduction of the initial registration fee from £25 to £15. Although the Council, in prescribing the annual fee, could differentiate between persons registered before or after a specified date,[2] as far as we are aware no such differentiation has ever been made. In the event, there are now two classes of medical practitioners on the register, viz. those who paid £25 and those who

1. *Report of the Committee of Federal Council on the Legislation Governing the Powers of the South African Medical and Dental Council and Other Matters Affecting the Relationship Between the Council and the Medical Profession*, this *Journal*, 9 August 1947, p. 577.

2. Section 95 (2) of the Medical, Dental and Pharmacy Act reads that: 'If at any time the funds of the Council . . . prove insufficient for its requirements, the Minister may, in his discretion, refund to the Council . . . any fees or allowances paid by the Council . . . in accordance with the rules framed under paragraph (c) of sub-section (2) of section *ninety-four* to members appointed by the Minister, or any other expenses necessarily incurred by the Council . . . in connection with such members, and further the Governor General, on the recommendation of the Council . . . may at any time by proclamation, authorize the Council to prescribe a fee to be paid annually to it by every registered medical practitioner. . . . In prescribing any such annual fee, the Council . . . may differentiate between persons according as to whether they have been registered before or after a date specified in the proclamation. . . .'

VAN DIE REDAKSIE

DIE JAARLIKSE HEFFING VAN DIE GENEESKUNDIGE RAAD

Van tyd tot tyd het die Federale Raad die geldigheid oorweeg van die Geneeskundige Raad se vereiste dat lede van die beroep, benewens die aanvangsregistrasiegeld, 'n jaarlikse betaling van £2 doen ten einde hulle name op die geneeskundige register te hou.

Die saak is inderdaad op die jongste vergadering van die Federale Raad geopper omdat baie van ons kollegas gemeen het dat dit nie 'n uitgemaakte saak is nie en die vereiste om hierdie jaarlikse betaling te doen, hinderlik gevind het.

Aangesien daar vooraf geen raadpleging met of kennisgewing aan die beroep was van die Raad se voorneme om 'n jaarlikse betaling te hef nie,[1] ontstaan die ergernis waarskynlik deels uit die wyse waarop die jaarlikse heffing ingestel is.

Die regsgeldigheid van die Raad se vereiste is van tyd tot tyd in sekere kringe in twyfel getrek en dit kan maklik wees dat die toedrag van sake tegnies nie bo alle twyfel is nie.

Ons weet wel dat kragtens art. 95 (2) van die *Wet op Geneeshere, Tandartse en Aptekers* die Minister die bevoegdheid het om kragtens art. 94 (2) (c) terugbetalings aan die Raad te doen. Ons weet nie of die Minister te eniger tyd sodanige terugbetalings gedoen het nie maar sulke terugbetalings sou in elk geval slegs die uiters beperkte uitgawe van vyf geneeskundige lede van die Raad gedek het.

Die jaarlikse heffing is vermoedelik opgelê omdat die Raad sy fondse moes vermeerder.[1, 2] Die instelling van 'n jaarlikse betaling is vergesel van 'n vermindering van die aanvangsregistrasiegeld van £25 tot £15. Alhoewel die Raad by die voorskryf van die jaarlikse betaling kon onderskei het tussen persone wat voor of na 'n vermelde datum geregistreer is,[2] is so ver ons weet, geen sodanige onderskeid gemaak nie. In elk geval, daar is nou twee soorte geneeshere op die register, nl. dié wat £25 en dié wat £15 betaal het as die aanvangs-

1. Hierdie *Tydskrif*, 9 Augustus 1947, bl. 577. *Report of the Committee of Federal Council on the Legislation Governing the Powers of the South African Medical and Dental Council and other Matters Affecting the Relationship Between the Council and the Medical Profession.*

2. Artikel 95 (2) van die Wet op Geneeshere, Tandartse en Aptekers lui: ‚As die gelde van die raad . . . te eniger tyd vir sy behoeftes onvoldoende blyk, mag die Minister volgens sy diskresie aan die raad . . . terugbetaal fooie of toelaes deur die Raad . . . ooreenkomstig reëls opgestel ingevolge paragraaf (c) van sub-artiekel (2) van artiekel *vier-en-negentig*, betaal aan lede deur die Minister aangestel of ander onkoste noodsaaklikerwys deur die raad . . . aangegaan in verband met sulke lede, en verder mag die Goewerneur-generaal te eniger tyd op aanbeveling van die raad . . . by proklamasie die raad magtig om 'n fooi vas te stel, deur elke geregistreerde geneesheer . . . aan die raad jaarliks te betaal . . . By die voorskrywe van so 'n jaarlikse fooi kan die raad . . . onderskei tussen persone na gelang hulle voor of na 'n in die proklamasie vasgestelde datum geregistreer is . . .'

paid £15 as the initial registration fee. Both categories, however, continue to pay the same annual levy.

It may, of course, be arguable whether the enabling provision in sec. 95 (2) entitles the Governor-General to proclaim the payment of an annual fee only at such times as the funds of the Council have proved insufficient for its requirements. The determination of this point becomes a legal subtlety and does not really affect the position of the practitioner who wishes his name kept upon the register.

Whatever the ethics of the procedure may be, in the form in which the Act was amended in 1946 there seems little doubt that, in order to have his name retained upon the register, a medical practitioner has no choice but to pay the annual levy, whether his initial fee was £25 or £15.

It may well be that more general approval of the Council's step in deciding to impose an annual levy would have been obtained if a differentiation had been made between persons registered before or after a specified date. The Council, however, saw fit, as far as we are aware, to make no such differentiation and it seems to us extremely unlikely that the validity of the annual fee can be challenged on any statutory grounds. The only likely basis for dispute hinges on whether the powers given to the Governor-General to authorize an annual fee can only be exercised if the funds of the Council prove insufficient for its requirements. As the section reads, the position may well be that Parliament has now delegated its powers in such a way that, once the annual fee has been proclaimed, the matter passes entirely into the control of the Council and the annual fee becomes independent of any such stipulation as may require proof of an insufficiency of funds for the work of the Council.

It is unlikely that any medical practitioner will be interested in challenging the various interpretations which may conceivably be placed upon sec. 95 (2), but we feel our colleagues should be reassured on the point that the annual fee is imposed lawfully and that the Council is entitled to erase from the register any practitioner failing or refusing to pay such a fee.[3]

3. Section 17 (1) (d) of the Medical, Dental and Pharmacy Act No. 13 of 1928 as amended.

registrasiegeld. Albei klasse gaan egter voort om dieselfde jaarlikse heffing te betaal.

Dit is natuurlik betwisbaar of die magtigingsbepaling in art. 95 (2) die Goewerneur-generaal die reg gee om die betaling van 'n jaarlikse bedrag af te kondig slegs op dié tye wanneer die fondse van die Raad ontoereikend vir sy behoeftes geblyk het. Die beslegting van hierdie kwessie word 'n taamlike ingewikkelde regsprobleem en raak nie werklik die geneesheer wat sy naam op die register wil hou nie.

Wat die etiek van die handelwyse ook al mag wees, in die vorm van die Wet soos dit in 1946 gewysig is, skyn daar min twyfel te wees dat, ten einde sy naam op die register te hou, daar vir 'n geneesheer niks anders te doen staan nie as om die jaarlikse heffing te betaal, of sy aanvangsgeld nou £25 of £15 was.

Dit mag wees dat die Raad se besluit om 'n jaarlikse heffing op te lê meer algemene goedkeuring sou weggedra het indien daar onderskei is tussen persone wat voor of na 'n vermelde datum geregistreer het. Die Raad het egter, so ver ons weet, dit dienstig geag om geen sodanige onderskeid te maak nie en dit lyk vir ons uiters onwaarskynlik dat die geldigheid van die jaarlikse betaling om statutêre redes betwis kan word. Die enigste waarskynlike grondslag vir geskil berus by die kwessie of die bevoegdhede wat aan die Goewerneur-generaal toegeken is om die heffing van 'n jaarlikse betaling te magtig, slegs uitgeoefen kan word indien die fondse van die Raad ontoereikend vir sy behoeftes blyk. Soos die artikel lui, kan dit wees dat die Parlement nou sy bevoegdhede op so 'n wyse gedelegeer het dat as die jaarlikse betaling eers afgekondig is, die saak volkome onder beheer van die Raad kom en die jaarlikse betaling nie meer in verband staan met enige sodanige bepaling wat bewys verg dat daar ontoereikende fondse vir die werk van die Raad is nie.

Dit is onwaarskynlik dat enige geneesheer belang sal stel in die betwisting van die verskillende vertolkings wat moontlik aan art. 95 (2) gegee kan word, maar ons meen dat ons kollegas bevestiging moet kry van die feit dat die jaarlikse betaling wettig gehef word en dat die Raad geregtig is om die naam van enige geneesheer wat versuim of weier om so 'n betaling te doen, van die register te skrap.[3]

3. Artikel 17 (1) (d) van die Wet op Geneeshere, Tandartse en Aptekers Nr. 13 van 1928 soos gewysig.

PERIARTERITIS NODOSA

REPORT OF A CASE WITH A REVIEW OF THE DISEASE

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The patient, 42 years old, with no history of any serious illness, a resident of Belgian Congo, developed what was considered, at first, influenza in August 1949. He had a high temperature and after eight days developed pains in his knees, which lasted a few days. He then noticed a red patch on his left foot. He received penicillin and later streptomycin treatment, which did not have any effect on the progress of his condition. A few weeks later he developed backache and then appeared a very important symptom, extreme weakness of his arms accompanied by pain, as well as severe pain in his lower extremities. He then noticed

small nodules on his forearm, which disappeared in 24 hours. His condition deteriorated, and he decided to go to the Cape, where he was hospitalized in the Groote Schuur Hospital. The following examinations were carried out:—

Blood eosinophilia, 8%; lumbar puncture revealed nothing abnormal; an electrocardiogram showed no evidence of coronary involvement; the lungs were normal, Wasserman and Kahn tests were negative, and the stools did not reveal any pathological organisms or parasites. Blood and urine were normal. There was no evidence of malaria in the blood. Biopsy of the unobtrusive subcutaneous nodules revealed the histology of periarteritis nodosa.

When I saw the patient, he appeared very tired, complaining of severe pain in his legs and of extreme fatigue. The pain in his arms had disappeared, and the use of the arm had returned. Clinically there was no involvement of any internal organ, and the blood and urine examinations were negative.

Comment. This case presents some features which do not fall in the usual pattern of the disease. The onset of the disease was ushered in by a fever incident, with myalgia, arthralgia and headache, to be followed later on by severe neuritic pains in both arms and later both legs, presenting a symmetrical polyneuritis. The finding of eosinophilia together with subcutaneous nodules lasting 24 hours, and resembling an urticarial phenomenon, in the later stages of the illness, may suggest an allergic etiology of periarteritis nodosa in this case. The clinical absence of any internal organ involvement, and the fact that the pain in the arms and weakness have disappeared, suggest that after the initial arterial insult, the disease has now regressed.

We can now formulate the positive diagnosis: *Periarteritis nodosa of symmetrical polyneuritic type without visceral involvement in its chronic regressing form.*

Etiology. The etiology of this disease has not as yet been established. Some believe that there is a specific infection, that the disease results from a hypersensitivity response of the vascular system to various antigens. There is an allergic factor, strongly supported by Rich[1] and cases of periarteritis nodosa have been associated with asthma, urticaria and angioneurotic oedema.

Miller[2] suggests that periarteritis nodosa is an allergic response of anaphylactic type to a variety of antigenic agents but not, invariably, bacterial in nature. The disease follows a recognized infection or drug intoxication. Miller dogmatically concludes that periarteritis nodosa is a chronic urticaria of the blood vessels, leading ultimately to arterial necrosis. Rich and Gregory[3] provoked an arterial necrosis in rabbits after injecting foreign serum.

Pathogenesis. Periarteritis nodosa enters into the group of diffuse vascular or collagen diseases, which include rheumatoid arthritis, rheumatic fever, disseminated lupus erythematosus, sclerodermia, verrucous endocarditis of the type Libman-Sacks, thromboangiitis obliterans, dermatomyositis. There is a striking similarity in their pathology. Yardumian and Kleinerman[4] present a very interesting study of the pathogenesis of collagen diseases and suggest that there is a resemblance pathologically between lesions found in them and those found in old age. In these diseases one observes that the ageing process becomes accelerated.

This process of accelerated ageing of the vessels forms the basis of the pathogenesis of diffuse vascular or collagen diseases. The writers describe various stages of this arterial insult. In the early stages there is infiltration in the adventitia and the disease is insidious with remissions and exacerbations.

The end stage is characterized by:

1. Fibroblastic degeneration;
2. Collagenous deposits;
3. Fibrinoid degeneration.

In periarteritis nodosa one can observe in the same patient simultaneously various stages from the inflammatory ones to the final necrosis of the arteries. The state of the arteries and the progress of the disease depends on the intensity of the stimulus and the number of remissions allowing healing. The fundamental pathology of the blood vessels changes has a direct relation to the type of exciting cause, to the intensity and consistency of the offending agent or agents, and to the response of each individual person.

The clinical picture of the disease will reflect the pathological state, which will shift from the mild intercellular infiltration to the other extreme of grave arterial necrosis. Thus the collagenous diseases and old age have a common pathology. In the former the process of ageing is accelerated and rapidly progressive; in the latter it is slow and accumulative.

Clinical Observations. Lovshon and Kernohan[5] report on 28 cases of periarteritis nodosa with peripheral neuritis. All extremities were involved in 10 cases. Involvement of a single nerve was not observed in any patient. The writers believe that 52% of all patients. present neuritis clinically.

Diaz-Rivera and Miller[6] report seven cases, five males and two females, the average is being 23.8 years, but in one case the disease occurred in an infant aged nine months. One patient had eosinophilia, and in six cases the onset was rapid with fever and tachycardia, dyspnoea and neurological changes were the outstanding clinical symptoms. The illness was of short duration and the majority of the patients died suddenly.

Logue and Mullins[7] present 11 cases and emphasize that it is more frequent among males than females. Miller and Daley[8] stress the fact that the diagnosis can be made during life and that rather more than 50% of patients recover. Becker and Chatgidakis[8a] report two cases of periarteritis nodosa observed in Johannesburg in the last 12 years amongst thousands of autopsies and emphasize that these cases were unascertained during life. In both the clinical symptoms were obscure, although in one case the diagnosis should certainly have been considered clinically, as it exhibited features, such as polymyositis, skin nodule, weakness, loss of weight and hypertension.

Selzer and Horwitz[8b] present a very interesting and well documented article on polyarteritis nodosa and describe seven cases observed, three diagnosed at

autopsy and four clinically. They discuss the etiology, stress the probable allergic basis and draw attention to the relationship between polyarteritis nodosa, rheumatic fever and glomerulonephritis. The writers suggest anti-histamine treatment.

Klein[8c] reports two unusual cases in which recovery took place. One had two histologically proved episodes of periarteritis nodosa separated by an interval of 12 years. The second patient had localized periarteritis of the appendix and was well 11 years after his discharge. The writer states that recovery may be expected if the disease is confined to predominantly non-vital structures, where there is minimal involvement of organs followed by healing and the localization of the disease to part of a single organ.

Rich[8d] reported the lesions of periarteritis nodosa in patients who died of infectious diseases, but in whose cases he demonstrated ante-mortem sensitivity to serum, sulphonamides, iodine. He was also able to demonstrate experimentally arterial necrosis by injecting allergens into rabbits. Following on the original new concept in histo-pathology devised by Klemperer, Pollack and Baehr[8f] under the name of "Collagen Disease", Cazal[8g] presents in a most interesting monograph his concept of "histiomonocytic reticulosis" (RHM) and groups a large variety of pathological conditions, such as Hodgkin's disease, lipoidoses, mycosis fungoides, eosinophilia granuloma, scleroderma and so on, some of which closely resemble collagen disease. This group of reticulosis would be the result of acute or chronic irritation of the reticulo-endothelial system and according to Gillman *et al*[8h] ramifications of the irritation of the reticulo-endothelial system are greater than suggested by Cazal and may include those syndromes generally regarded as having a rheumatic origin and associated with cardio-vascular system and with the kidneys. Gillman *et al* were successful by using trypan blue in rats to reproduce almost all the pathological pictures included in Cazal's concept of histiomonocytic reticulosis (RHM). There seems therefore reason to suppose that some correlation exists between collagen disease of Klemperer and Cazal's reticulosis.

Periarteritis nodosa belongs to the group of diffuse vascular or collagen diseases and *there seems to be some relation with another group of diseases, of histiomonocytic reticulosis type.*

Bonnin *et al.*[9] present a number of cases of periarteritis nodosa, and stress that subcutaneous nodules are the most characteristic elements of that disease, but are absent in a good many cases and may be so until the terminal stages of the acute or sub-acute forms, appearing only in the chronic forms. These authors suggest that any protracted fever, with haematuria, anaemia and muscular or joint pains, must arouse suspicion of periarteritis. From the therapeutic point of view, these authors claim to have obtained good results with antihistamine and penicillin therapy.

Langeron *et al.*[10] report clinical observation of a Fiessinger-Leroy syndrome (conjunctival-urethral-synovial syndrome) with subcutaneous nodules and periarteritis, and discuss the etiology and pathogenesis of mild and curable forms of periarteritis nodosa.

Griesbacher[11] reports on four cases and emphasizes that periarteritis provokes peripheral neuritis, epigastric pain, haematuria and fever with subcutaneous nodules and hypertension. He states that in all his cases sternal puncture revealed an early eosinophilia, yet this was absent or insignificant in the blood. There was also an augmentation of the reticulogen count and a diminution of lymphocytes. According to this writer, penicillin brought the fever down and the sedimentation rate was lowered. He also suggests an allergic etiology of this disease.

Clinical Picture. Now that more attention is being drawn to the frequency of this disease, diverse clinical types can be differentiated:—

1. *Acute Forms.* From mild forms, such as described by Miller as *formes frustes*, to extremely severe ones.
2. *Subacute Forms.*
3. *Chronic Forms*: (*a*) *Visceral Type*, depending on the organ or organs involved.

(*b*) *Neuritic Form*:—

i. *Asymmetrical Type.* Asymmetrical mononeuritis simplex, affecting one solitary nerve (Miller[2]).

ii. *Symmetrical Type.* Symmetrical polyneuritis associated with the asymmetrical one.

iii. *Symmetrical Type.* Symmetrical polyneuritis, unassociated with the asymmetrical type.

(*c*) *Viscero-neuritic Form.*

In the acute and subacute stages the disease is ushered in with fever (which may be protracted) myalgia, arthralgia, leucocytosis (20,000 to 30,000 per c.mm.) raised sedimentation rate, severe headache, tachycardia. Urinary symptoms such as haematuria, albumin and casts may appear early in the illness.

In the chronic forms the symptomatology becomes completely disorganized and confused. Bizarre symptoms appear indicating the involvement of internal organs.

Diagnosis. This no longer depends on autopsy findings, nor should the disease be considered rare and fatal. When attention is drawn to the various manifestations of this illness, it will be easier to find diverse clinical forms of this disease. If post-mortem examinations would be directed to the state of the vascular system, one would be surprised to see how frequently the characteristic vascular changes are found in many patients never suspected of periarteritis nodosa.

The following salient points help towards a positive diagnosis:—

1. *Myalgia, Arthralgia* and *Neuritis*, with protracted fever and weakness.
2. *Peripheral Neuritis* with fever, with or without erythematous patches and/or subcutaneous nodules.
3. *Paraesthesia, Wasting and Muscular Weakness of a Limb*, following an acute illness. To be differentiated from certain organic neurological conditions.
4. *Any Obscure Disease in which Neuritis Appears.*
5. *Abdominal Pain*, associated with acute rheumatism, so-called 'abdominal rheumatism' or any other abdominal pain often confused with appendicitis, cholecystitis, should arouse suspicion of abdominal periarteritis nodosa.
6. *Rheumatic Fever* in a surgical ward, like

abdominal pain, may be a manifestation of arterial necrosis (Spiegel[12]).

7. *Hypertension*, especially in young patients, associated with fever and neuritis.

8. Any new symptoms such as muscular pain or weakness, neuritic pain, appearing during a course of a drug treatment, must arouse suspicion of periarteritis nodosa, as a result of drug sensitization. (A great variety of drugs has been suggested as liable to cause arterial necrosis, e.g. sulphonomides, aspirin, iodine, dilantin, epanutin, organic arsenical preparations.) Van Wyk and Hoffman[14] report a fatal case of periarteritis nodosa resulting from sodium dilantin sensitization.

9. *Nodular Vasculitis*. This condition can easily be differentiated from the subcutaneous nodules of periarteritis nodosa. The nodules are larger, resemble most closely those of erythema nodosum, being dusky red and mildly painful. Histologically, they show only thickening of arterial walls with swollen endothelial cells, but there is no evidence of polymorphonuclear infiltration, or arterial necrosis, as seen in periarteritis nodosa. Woodburne and Philpott[13] give a detailed description of nodular vasculitis and emphasize that the condition is benign.

Prognosis. To-day the prognosis should be revised. As we are able to differentiate clinically various forms of periarteritis nodosa, from the mild to the severe ones, from the acute to the chronic types, we begin better to understand the underlying pathology and learn that the prognosis depends in direct proportion to the degree and extent of arterial lesions inflicted. Sometimes the lesion can be reversed and the disease arrested.

We can assume that a number of mild cases, unascertained clinically, fall into the group of the mild types which have recovered and which were diagnosed as arthritis, neuritis and other such clinical labels. The prognosis is not fatal, a good many cases recover, and only those which suffer multiple visceral arterial insults succumb.

Treatment. No specific treatment has as yet been proposed for this disease. Some suggest antihistaminic treatment, others penicillin therapy. Both these have been tried on the present patient, without, however, any improvement. Since the work of Hench *et al.*[15] on the treatment of rheumatoid arthritis with certain steroids of this adrenal cortex, many American writers (Elkinton *et al.*[16]) used the adrenocortioctrophic hormone (ACTH) in some collagenous diseases, such as rheumatoid arthritis, disseminated lupus erythematosus, dermatomyositis, acute rheumatic fever and status asthamticus. These writers as well as others (Thorn *et al.*[17]) have reported encouraging results from the use of these adrenal steroids.

We have been induced to apply this treatment, as periarteritis nodosa falls in the group of collagen diseases. Unable to procure ACTH in this country, we have given a substitute in the form of deoxycortone acetate (DOCA), coupled with ascorbic acid. The patient received daily injections of 15 mg. DOCA. The pain in the legs appeared to be eased, and there was a feeling of remission of the neuritic attacks. The patient has now left for the Belgian Congo, where he will receive ACTH.

Cases have been reported where encephalopathy has resulted from this medication with adrenal steroids and hypophyseal corticotrophic hormones. Also metabolic changes indicate that potassium deficiency may result as well as retention of sodium, causing oedema. One must, therefore, undertake this new and fascinating therapy with some caution. So far it has not been possible to establish the rationale of this treatment, nor explain how these steroids affect the course of collagenous diseases.

Very recently Zarafonetis *et al.*[18] reported some good results with PAS (para-amino salycilic acid) in the treatment of diffuse lupus erythematosus, dermatomyositis and scleroderma, which fall into the group of collagen diseases, although they admit that further investigations are warranted. It may, therefore, be suggested that alongside the adrenal steroids, PAS should be given a trial with ACTH.

CONCLUSIONS

1. An unusual case of periarteritis nodosa is presented, which began as an 'influenza' incident and later as a symmetrical polyneuritis, with subcutaneous nodules and without visceral involvement.

2. It is suggested that mild forms of this disease have so far escaped our notice and that such cases recovered and were diagnosed as myalgia, neuritis, arthritis, etc.

3. Allergic, bacterial and infectious factors enter into the etiology of this disease.

4. Pathologically, it belongs to the group of diffuse vascular or collagen diseases, where the accelerating ageing process plays a predominant part.

5. Treatment with adrenal steroids coupled with PAS should be attempted, whilst anti-histamine therapy should also be envisaged and ACTH.

6. The prognosis of this disease should be revised and one should be able to diagnose and differentiate various clinical forms of this condition.

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REHABILITATION OF THE INDUSTRIAL WORKER*

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Until recent times, industry in this country has had on the whole sufficient labour for its requirements, and the need to conserve its manpower, or maintain it at a high degree of efficiency has never become apparent. The wastage due to sickness or injury has been accepted as inevitable, little thought or attention being given to expediting the return to work of the sick and injured or to employing to the fullest extent of their capacity those who are handicapped in some respect or other.

'If we are confronted with an industrial health problem, the first question we ask is "What are the facts?" and the next, "Are these all the facts?" Then almost invariably we find that although the problem is inherently medical, the chemist, the physicist, the management or the workers, the psychologist or some other experts have vital roles to play in solving the problem'. (Merewether.)

Rehabilitation is not only a medical but also a sociological problem, and in those countries where the productive effort of each individual is essential to the well-being of the nation, a new attitude has developed towards the restoration to health, working capacity and social independence of the incapacitated. This approach to the subject has led to the establishment of fracture clinics, occupational therapy and vocational training centres, retraining workshops, and rehabilitation centres for the industrially fatigued and maladjusted.

Into these various schemes have been enlisted the services of surgeons, general practitioners, psychiatrists, industrialists, social welfare workers, industrial psychologists, disablement rehabilitation officers, occupational and physio-therapists, motion study engineers and others whose aim it is to refit the worker for his ordinary work or, if this is not possible, to train him for work in another occupation.

'The practice of rehabilitation for any doctor begins with the belief in the basic philosophy that the doctor's responsibility does not end when the acute illness is ended or surgery is completed; it ends only when the patient is trained to live and work with what is left. This basic concept of the doctor's responsibility can be achieved only if rehabilitation is considered an integral part of medical service'. (Rusk.)

The importance that is being attached to this field of medical practice is shown by the extent to which rehabilitation has entered into the future planning in New York City in both private and public hospitals. In the report of the Hospital Council of greater New York it was suggested that 25% of the city's general hospitals should be allocated for convalescence and rehabilitation. This would mean one such bed for each 1,000 of the city's population.

Although this approach to the needs of the disabled and handicapped has received practical acceptance in a number of countries, the principles, apart from some military units during the war and the sheltered employment depots for ex-Service personnel, have not as yet been generally applied in South Africa.

With the rapid industrial development that is taking place, and the increasingly apparent shortage of skilled labour, the need for the retraining and re-abling of the incapacitated worker is becoming increasingly urgent.

METHODS OF REHABILITATION

Early Treatment and Return To Work. It has been said that rehabilitation should start at the moment of injury. Whilst this may possibly be considered an exaggeration, it is nevertheless true that early treatment, both from the physical and psychological standpoints, does much to reduce the period of disability. It is a great advantage to keep the worker at work, and every endeavour should be made to do so. With the enlightened co-operation of the management, and some ingenuity on the part of the doctor, it is remarkable to what extent the continued employment of the injured workman can be achieved. Fears of ill effects having been allayed, and the necessary amendment made in respect of duties, the workman is, as a rule, only too ready to remain on the job. Thus the problem of rehabilitation is minimised or does not arise. One is often struck with the long periods of needless absence of men who remain away from work on the sincere advice of those undertaking their care. Unfamiliarity with work processes, or the practicability of making adjustments to allow for the disablement, influence the doctor to 'play safe', to the detriment both of the worker and production.

'It is well recognised that there are certain therapeutic indications for rest, but these indications are far less common than has been believed in the past. This leads us to review all injuries, to reduce immobilisation to the

* This paper was read at the Medical Congress of the Association held at Cape Town in September 1949.

minimum and to encourage function in all structures that do not demand rest'. (A. R. Thompson.)

This point of view is well illustrated in the treatment of injuries to the hand. Having immobilised any fracture, or, in the case of infections, as soon as the acute stage has subsided or a felon drained, it has been our practice to keep all parts that are not injured unrestrained and actively to encourage the use of the limb as a whole. In this way we have largely avoided stiffened finger joints so often resulting in permanent limitation of movement, or at best, taking many weeks to correct. One should mention that re-education of a fractured digit released from immobilisation requires close supervision. It is at this time that the worker's duties must be most carefully selected.

Psychological and Personal Factors. 'If medical practitioners were as much alive to the psychological as to the physical dangers of a patient who has been in an accident, if they took as much care to prevent infection of his mind with insidious ideas as they did to prevent infection of his body, many of these cases would be cleared up'. (Farquhar Buzzard.)

The bodily shock associated with serious injury has its counterpart in the mental shock of a man suddenly confronted with the economic and social consequences of his disablement. He is beset with worries and problems, some real, some imaginary, concerning his family and his job. 'There is a lot said about the psychological sequelae of accidents and that the patient gets emotionally disturbed as a result of his accident. I think we feel that a good deal of this is not so much the direct result of the injury, as it is of anxiety as to what is to be the economic sequel to the accident. Is he going to be permanently disabled? Is he able to carry on financially until he gets back to work? What is going to be the economic effect in general among his family?' (L. P. Lochart.)

It is important to recognise the emotional trauma at an early stage, and to prevent the fears and apprehensions from becoming fixed. The emotional problem, if not discovered and treated may become a worse disability than the physical injury. It is here that the general practitioner and the industrial medical officer, who is known to the patient, can be of great service. He can explain the nature and extent of the injuries, instil courage, interpret the meaning of the various procedures and indicate in terms that the patient can understand, the medical, vocational and economic effects of his injury.

Difficulties due to reduced income or lack of paternal control need the attention of someone experienced in these matters. In all cases where prolonged absence from home is anticipated, investigation by a welfare worker is indicated, in order that his or her assistance in the prevention and solution of such problems may be forthcoming.

Interest and encouragement on the part of the employer, or a responsible representative, is of major value. It is not enough to offer vague reassurances that everything will be looked after, and that there is no need to worry. A programme should be drawn up and the goal defined as soon after the injury as possible. In the planning of this programme, the patient's role should be an active one. Guidance by others should be readily available, but he should to a large extent be responsible for all major decisions. It is necessary to study the patient from every aspect; his educational standards, his mental make-up, his aptitudes and inclinations should all be assessed and form the framework upon which the programme is planned.

Nor must the patient's hobbies or sporting activities be forgotten. The injury may be such that, whilst the job may be little affected, the possibility that he will be prevented from participating in some recreation or hobby will militate against his recovery.

In a certain number of cases where the worker's occupation is of a hazardous nature an anxiety state may develop having its origin in an averseness to returning to work with the consequent risk of further accidents. This condition is frequently of a temporary nature, and suitable treatment during the period of incapacity may clear it up. When, however, there appears to be a likelihood of a real difficulty in the resumption of his employment, training for an alternative vocation may prove necessary.

A small but troublesome group of cases consists of those persons who are constitutionally predisposed to functional nervous disorders. Any untoward event or adverse circumstance in their lives is prone to upset their mental balance and precipitate a neurosis. The psychological reaction bears little relationship to the severity of the accident, and may complicate a trifling injury to an extent out of all proportion to its physical significance. These cases often prove difficult of treatment, trying the patience of all concerned.

The early recognition of such emotional imbalance, together with the expert assistance of a psychiatrist, does much to bring the disorder under control.

AFTERCARE AND REPLACEMENT

'It is not enough to get a man back on his legs; he must be got back to his job, and this entails the prolongation of treatment beyond the hospital stage'. (F. G. Ward.)

It must be admitted that this country is seriously lacking in rehabilitation facilities. Through this lack patients are being discharged from hospitals to their homes, to make their way as best they can, either to their old employment, or, if in their opinion or that of their employers this is beyond their altered capacities, to some alternative job which they hope they will be able to perform. In most instances a certificate stating their inability to do more than 'light work' is the sum total of the guidance of which they are in receipt.

Whilst in the great proportion of cases the conclusion of surgical treatment, followed possibly by a short period of physio-therapy and exercises, finds the patien table to return to work, there remain a considerable number of patients, who at the end of this stage, are not yet fit to return to their normal work—either because the hospital is not provided with the equipment for physio-therapy and remedial exercises for completing the full restoration of functional activity, or because the man's normal work is of so arduous a character that a general reconditioning is needed as well, to fit him for it.

In the absence of further care they languish in their homes, unhappy and depressed; often ill-nourished owing

to reduced income, with little or nothing with which to occupy their time. At best they make a protracted recovery.

Frequently their skills are lost to them, and since no functional analysis or retraining is available, they eventually find themselves relegated to the dust heap of the permanently crippled or unemployable.

The need is to fill the gap between leaving hospital and returning to work, to provide an alternative to the annoyance and irritation of an overcrowded home, to furnish a means of reconditioning for those who are not equal to the strains involved in their normal work. Where it will not be possible for the workman to resume his former occupation it will be necessary to assess his capacity and to train him to make the most of what functional ability remains.

The usual attempt to solve the problem is by means of the provision of 'light work' at the workman's place of employment. Very often this is successful, the man gradually acquiring his full bodily strength. Not only are many employers eager to assist in this way, but often the man's own workmates will help him along through the period of disablement.

On the other hand, the receipt by the employer of a certificate recommending light work may prove nothing but an embarrassment. The nature of the industry may make it impossible to carry it out, or, the rate of pay for the modified duty being less than the compensation rate, the workman may be unwilling to avail himself of it.

Nevertheless the provision of light work is a most useful method of rehabilitation, and some firms have selected particular jobs for those in need of special consideration. There are, however, certain dangers in the haphazard assignment of workers to these duties. Usually the approach is a negative one; that is to say, the job chosen is one in which use of the injured part is avoided. Whereas graduated activity of the part is the principle underlying modern methods of rehabilitation.

Thus, unless arrangements are made for the continued medical supervision of the workman, his progress will lie either within the uninformed discretion of his foreman, or be determined by his own feelings in the matter. As a result he may choose to undertake more than he is capable of doing, or through lack of a progressive scheme, will accept light duty as his permanent lot.

Some large overseas firms, having realised the importance of this matter, have made elaborate arrangements for the rehabilitation of their sick and injured employees. The policy is usually to return the workman if at all possible to his own department, or at any rate to work within the factory. The stimulus to morale is obvious.

For those for whom such disposal is not desirable, rehabilitation workshops attached to the factory are provided. There, within their own working environment the necessary remedial measures to bring about his rapid recovery are instituted.

The basic principle of these schemes is the value of rhythmic, repetitive movement in the manufacture of standard production components. Through the medium of machine and bench work or light assembly, modified and adapted to secure the necessary movements, remedial exercises, prescribed by the works' medical officer or the visiting orthopaedic surgeon are carried out. The introduction of the specialist surgeon into the industrial environment is an interesting and important feature of the scheme. Forming part of a team consisting of the works' medical officer, the rehabilitation officer and the factory supervisor, he prescribes the necessary graded work or remedial exercises, advises on the mechanical adaptation of machinery for this purpose, assists in the placement of handicapped personnel, and maintains supervision over his cases until they are restored to full function.

These rehabilitation workshops, although originally intended for accident cases, have developed along general lines, and are usually made available for the treatment of all types of incapacity, whether surgical, medical or psychological. In some units physio-therapy and physical training facilities are incorporated in the scheme, and opportunities for vocational training may be provided.

The system by which workmen are rehabilitated in workshops attached to their own factories, although very satisfactory, can be applied only in the case of firms employing a large number of workmen, and where the type of manufacture can be adapted to remedial purposes.

Owing to the industrial conditions obtaining in South Africa, its application here would of necessity be very limited. It would appear that the rehabilitation requirements of the country might best be met by group schemes, based on, but not necessarily a part, of the existing hospital services.

GENERAL CONSIDERATIONS OF REHABILITATION

The arrangements for the establishment of rehabilitation services will necessarily vary according to the circumstances and needs of the areas in which they are to be situated, but there are certain features common to all types of unit. These may be considered under the following headings.

Psychological. From the psychological standpoint the first essential is the stimulation of enthusiasm and willingness on the part of the patient in his participation in the activities of the unit. This requires an optimistic, cheerful, sympathetic attitude on the part of the staff, who must be capable of conveying their own confidence to the patient. Whilst it is necessary to create in him the conviction that everything will be done to return him to work, he must nevertheless be made to feel that his own endeavour is essential to effect progress.

There are workers in this field who consider that the rehabilitation unit should always be geographically separate from the hospital and its associated atmosphere of morbidity. Functionally, however, they should be closely allied in all their activities.

In some schemes the factory has been brought within the walls of the hospital, and working in familiar conditions, the man is 'rehabilitated for his trade in his trade'. By means of a period of work at his normal job his ability and his willingness to return to his trade is assessed.

Physical. The physical basis of restoration of function is founded on the principle of active, graduated exercise.

These exercises, designed both to maintain the general bodily tone and restore the function of the injured part, are instituted early in hospital treatment, and progress gradually on well-established physio-therapeutic lines to the remedial exercise and physical training departments.

In this way is attained the full 'toning up' necessary to enable the patient to withstand the stresses and strains of heavy manual work, and the functional recovery not provided by the surgical treatment in hospital.

Group exercises and games, through the stimulation of the competitive instincts, and the elimination of the fatigue and boredom attendant upon set exercises, form a valuable method in the attack on the injury.

As mentioned earlier, the reconditioning necessary may be obtained by means of suitable light work at the worker's place of employment. In such cases a system of organised placement and supervision should be an extension of the function of the unit.

Social. The role of the welfare or social worker attached to the rehabilitation unit is a most important one. His or her duties embrace the whole sphere of the patient's activities; they include the investigation and smoothing out of domestic difficulties, personal contact with the employer to assist in his replacement in industry, and the maintenance of the patient's interest in his cure.

As a member of the team he will be familiar with the physical and mental condition of the patient; this knowledge together with his appreciation of its social and industrial implications enables him to see the problem of replacement in its true perspective. Through him the liaison between the rehabilitation centre and the employer is largely effected.

Functional Analysis. Before placing a man back in industry it is necessary to assess his capacity for work. It is more than saying he can bend his back so far, or flex his arm so many degrees. It is essential to know what actual jobs he can do with what amount of movement, what substitute functions he is likely to acquire, in what form his temperamental make-up or educational qualifications will permit of retraining.

The determination of a man's working capacity requires the collaborative effort of the whole rehabilitation team, each member constantly bearing in mind the ultimate aim of rehabilitation—that of fitting the man into his industrial environment. It implies a knowledge of work processes and an understanding of the physical and psychological demands of the operation. It will need, too, the co-operation of the industrialist, who through the work of the motion study engineer and the industrial psychologist, complementing that of the anatomist, physiologist and surgeon, will furnish the job analyses upon which placement recommendations can be made. The importance of the part industry has to play in discovering what job an injured man can do with greatest benefit to himself and his employer has repeatedly been stressed. It is to the industrialist that the medical man turns for information which is as necessary for the completion of his task as his splints are at its commencement.

The Rehabilitation Team. The existence of a rehabilitation team has been implied several times in this paper. We have mentioned the physio-therapist, the welfare officer, the surgeon, the occupational therapist, and so on. Every team, however, must have its activities co-ordinated—some one person must be responsible for the smooth running of the whole. This responsibility—the responsibility for effecting the complete rehabilitation of the patient must remain that of the surgeon, and it is felt that it is he who must accept the position of leadership in a team working essentially in concert.

CONCLUSION

Enough has been said to indicate the necessity for the institution of a rehabilitation service in South Africa. What form it should take, how it is to be financed, details of administration and staffing are outside the scope of this paper.

It is felt, however, that these are matters long overdue for study and investigation.

There seems to be a large and fertile field for research, not only into the question of rehabilitating the injured workman, but also into the problem posed by those who, whether through sickness or physical defect, neurosis or emotional disorder due to maladjustment at work, are unable to contribute efficiently to the productive effort of the community.

SUMMARY

There is a growing awareness in other countries of the need to conserve and use to its full capacity the manpower of the nation. In South Africa we have accepted the wastage due to sickness and injury as inevitable. It is pointed out that, owing to the rapid industrialisation of the country and the shortage of skilled personnel in our labour force, we cannot as a nation persist in the present apathetic policy—this apart from humanitarian considerations.

The importance of the early treatment of injuries and the advantages to be gained by, when possible, keeping the workman at work, are stressed.

Associated with physical injury are psychological disturbances having their origin in the fears and apprehensions regarding security of employment, economic difficulties affecting the family and the possibility of permanent disability. Psychological factors are present in varying degrees, but if not recognised and treated, militate against recovery.

Often it is the practice to leave to the patient the task of regaining functional activity after surgical treatment is completed. The absence of facilities for aftercare may result in prolonged periods of disability or permanent loss of function.

By various rehabilitation methods—occupational therapy, physio-therapy, remedial exercises and the assignment of supervised light duty in the factory—the gap between leaving the hospital and the resumption of normal work may be bridged.

Retraining workshops, developed by certain large overseas firms, in which remedial exercises to restore function are achieved through the adaptation of machinery, are described.

The rapid restoration to full working efficiency and the retraining and resettlement of the permanently disabled is the work of a team functioning as an integrated unit. An essential member of this team is the industrialist.

Although no rehabilitation scheme for application in this country is detailed, the urgent need of such a service is emphasised. Attention is drawn to the desirability of investigation and research into the whole problem.

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COMMON MEDICAL SYNDROMES IN PHYSICAL EXERTION*

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In this age when sport, often in its most violent forms, is a strong feature of everyday life, it has become common for the medical man to be asked for advice about the effects upon the human frame and heart. It is not easy for a doctor to get access to enlightened information on the subject. Such writings as there are, are scattered, difficult of access and often extraordinarily prejudiced and lacking in cool scientific deduction. It may be of service then, to discuss in general outline, such aspects of the problem as may commonly be put before us. The simpler syndromes will be given first for the sake of clarity.

1. *Athlete's Sickness: Description.* It occurs only after rapid violent effort carried to the limit of human endeavour; slow prolonged effort will not cause it unless it terminates in maximal speed of effort. At the end of the effort, e.g., most typically at the end of a 440 yards' race, the limbs stiffen and cramp generally, and may be felt to be hard and hypertonic, probably due to the accumulation of waste products of muscle action, especially lactic acid. Due probably, to cerebral ischaemia, vision becomes slightly yellow, distorted, or even black. Fainting may occur but rarely does. Nausea ensues and persists with lassitude plus restlessness and a general rotten feeling and culminates in vomiting within some ten minutes; this may be repeated, and is followed by a feeling of well-being and return of energy. It is often regarded as being due to unfitness, which is not the cause. Naturally, the fitter a man, the less easy it is for him to provoke it as he is capable of greater and more sustained effort. Nevertheless, however fit a man is, he who is liable to it will get it if pushed at high speed to his maximal effort until the limbs will no longer respond. Cyanosis of lips, plus great facial pallor occurs. Skin circulation is cut down by arteriolar contraction.

Pathogenesis. It is due to the profound disturbance of blood chemistry. An oxygen debt of 18 litres may be accumulated and a tremendous degree of acidosis for, in the events which produce it, respiration is reduced almost to nil. In sprints, the epiglottis is held open but no respiratory effort is made. It is impossible to close the glottis more than momentarily because venous return to the chest is tremendous and impeded thereby; intrathoracic pressure rises if the glottis is closed, as abdominal and thoracic muscles are in contraction. Some air is jolted in and out of the chest but serves only to aerate the dead space of trachea and bronchi. CO_2, if it could be formed fast enough from the lactic acid, cannot be discharged. Lactate rises in the blood plasma, 10-200 mgm.%, as it cannot be part converted as normally into CO_2, H_2O and glycogen, etc., within the muscles where contraction under partially anaerobic conditions has been taking place.

(i) Adenosine triphosphate breaks down (exothermic) and reforms (endothermic).
(ii) Creatine phosphate breaks down (exothermic) and reforms (endothermic).
(iii) Glycogen-lactic acid (exothermic) to form lactic acid and is *not* reformed (hence the accumulation).

Further details will not be given here as they are involved. (S. Wright, *Applied Physiology*, 1945).

2. *Hyperpnoea and Alkalosis.* There is a common superstition that an O_2 reserve may be built up before taking part in an event by over-breathing and it is commonly practised. Due to the fact that the competitor is nervous and hyperadrenalised by the excitement of anticipation of competition, his circulation is much speeded up in the lungs and muscles. Hyperventilation is therefore particularly effective in evacuating CO_2 from

* This paper was read at the Medical Congress of the Association held at Cape Town in September 1949.

the lungs, H_2CO_2 in plasma falls, and alkalosis results with the typical syndrome now well known to physicians among neurotics and nervous individuals. Tingling begins in the ulnar distribution of both arms, spreads to the lips, tongue, face, and may become general to be succeeded by hypertonicity of all muscles so that eventually cramp ensues and the arms and hands adopt the accoucheur's position. Needless to say, the practice is harmful to efficiency by virtue of the hypertonicity of somatic muscles. The remedy is obvious.

3. *The Process of 'Warming Up', of 'Stitch' and of 'Second Wind'.* The practical necessity—for it is a good deal more than mere desirability—for taking exercise immediately prior to any event has long been recognised—but theory has lagged behind and widespread acknowledgement of it even further so. Injuries, especially to muscles, tendinous insertions and bursae occur with readiness to a 'cold' individual imposing strain upon himself muscularly—especially violent and sudden strain. The quiescent muscle, including the myocardium, has perhaps one blood capillary in ten open and conducting blood. Arterioles are mainly semi-contracted and circulation through them much below maximal. Exercise soon opens the remaining nine, or reserve, capillaries and arterioles dilate, and this is the essential change in 'warming up'. In addition to this the blood supply to the abdominal viscera is lessened. Adrenalin helps in both these processes. Adrenalin is secreted or rather allowed to enter the circulation in greater quantities in anger and excitement. It is responsible for the tremulousness of the hands, dilated pupils and general state of readiness (N.B. *not* fear) of the individual. It causes arise of glucose in the blood stream by action on the liver, a glycosuria results in some, and is quite harmless. It, and the excitement, are also, since I am on the subject, the causes of dry mouth and the almost nauseous feeling in the epigastrium. Diarrhoea occasionally occurs and frequency of urination without polyuria of corresponding degree, and these are more fear or anxiety signs, likewise palmar sweating, and are not so beneficial to the impending effort. An old trainer (McKerchar), once said to me by way of a hint—'always shake hands with your opponent; if his hands are sweating you have got him beat'. It's true if your own hands are dry! Fear and anxiety seem to be weakening and in extreme cases, paralysing emotions, and are most harmful to efficient physical endeavour.

'Stitch'—familiar to you all in boyhood, is said by Sir Thomas Lewis to be due to ischaemia of the diaphragm and this fits the known facts—the steady pain made worse by every inspiration, the situation of the pain on either side of the chest under the lower ribs, and its disappearance if the speed of effort is lessened to allow of dilatation of capillaries and release of muscle spasm by removal of waste metabolites. As is well known, it occurs early in a race, and especially easily in those who compete without 'warming up' and whose circulation therefore is not fully activated first. It conforms also with the fact that rest or slowing of pace is forced upon the competitor by mounting pain (probably identical with that of Angina Pectoris), for the spasm of muscle must compress the capillaries and arterioles and forbid their opening up further until effort is reduced. Then, probably, local reflexes and local chemical action take place encouraging blood vessel dilatation.

There is a second type according to Lewis, a deep seated precordial pain, constant and eased by rest and due to myocardial ischaemia and 'cramp'. To quote him exactly, because I cannot fully agree with him; 'the heart muscle (right ventricle) relaxes, there is diminished resistance and the increased intracardiac pressure acting against this diminished resistance causes dilatation of the cardiac chambers. The pain gradually disappears and the dilated heart develops a new power which enables it to perform an extraordinary task, free from pain just when all seems lost—this is "second wind"'. With deference I disagree with his supposition that this is 'second wind'. The phenomenon does not occur in a fully warmed up person even if he is unfit. His phraseology is difficult to follow. Second wind seems more probably to be due to dilatation of the reserve capillaries in heart and somatic musculature and the opening up of arterioles. Shortness of breath (and sometimes 'stitch') is an invariable antecedent to the coming of second wind and relief, and obviously there is, as would be expected, a sudden rise of H_2CO_3 in the plasma just before and during the early capillary dilatation. Once the reserve circulation is fully open, katabolic products are carried away as they form and do not accumulate as before in the muscles. That, at least, is a reasonable explanation though the physiologists state that the etiology of second wind is quite unknown. (S. Wright, Eighth Edition *Applied Physiology*).

4. *Hypoglycaemia.* This requires only a short word for most of us are familiar with the shakiness, irritability and loss of co-ordination that may occur during a game, which is transitory and which is due to a low blood sugar. The participant most liable to adrenalin effects prior to competing, is obviously most subject to it, as adrenalin has depleted his liver reserves. It therefore often affects the best performers. Even if glucose is not taken, it tends to pass off as more adrenalin is secreted and manages to get more glucose from the liver. It is clearly best to supply glucose between events. It does seem to me, however, that a case could be made out for endeavouring to adapt the body to doing without additional glucose in all except prolonged effort, as in five-set tennis events, for it appears that those who take glucose regularly, seem to become more and more dependent upon it and more liable to minor hypoglycaemic symptoms, perhaps due to an unduly fluctuating glucose blood level.

5. *Chloride and Water Metabolism.* This is a long subject which I must perforce abbreviate and oversimplify, as I am reviewing many conditions and do not want to dwell too long on any one of them. Haldane did the pioneer work and this was followed by a fascinating piece of research work by Talton when the Boulder Dam was being built in California. There have, of course been other excellent pieces of research, too many to mention (McCance *et al*).

The dehydration from sweating means loss to the body of NaCl and water; other chemicals are lost in minute quantities and have no known significance to performance. A great deal of sweat may be lost, 4 to

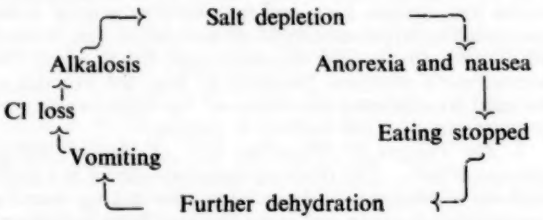
6 lb. body weight, during a day's cricket in a hot, humid climate is not uncommon. Tiredness and fatigue result, and thirst which is not proportionate to the amount of fluid sweated away. The consequence is that inadequate quantities are drunk in replacement. Extracellular dehydration occurs. If water without NaCl is drunk, electrolytes are withdrawn from the cells to compensate the extracellular dilution; this is a limited mechanism. Because there is a loss of extracellular osmotic pressure, the kidneys excrete water and extracellular fluid volume and plasma volume fall despite drinking of unsalted water, and further dehydration or secondary dehydration ensues. Acid-base distribution may be disturbed by loss of chloride, and alkalosis may ensue especially if there is vomiting. Below a certain level in the plasma, the kidneys will no longer excrete Na or Cl. (Sodium below 320-350 mgm.%, chlorine below 340-385 mgm.%.)

The effects are lassitude of two main types, depending on whether dehydration and water replacement without proportionate NaCl is acute or 'chronic'. Lassitude and headache are the chief indications of the latter. People who live in hot humid parts often suffer from this easily avoidable state of affairs and, I am sure, some cases of 'Natal fever' come under this heading. It accounts, probably, for the fatigue, irritability and early 'staleness' affecting some members of touring teams in hot humid weather. Some degree of acclimatisation occurs, but not enough. Inhabitants of hot countries secrete less NaCl in both sweat and urine, and those having hard physical work in similar conditions will, in the course of a few weeks, also start to conserve salt. It is more efficient, however, for athletes on tour, playing frequently and sweating heavily, to be given extra large quantities of salt and fluid.

Simple acute dehydration is rarely of sufficient degree to call for attention in athletes except boxers who force sweating to lose weight. Cellular dehydration ensues as fluid is drawn from here to make up blood volume, alkalaemia from chloride loss, fast pulse and exhaustion develop.

In the more acute episodes, exhaustion is more commonly followed by 'water cramps'. Severe cramp may come on and will always attack the same group of muscles in any individual. After the game, he starts to drink, and within a few minutes cramps begin, and may be agonisingly severe, or mild. This is the main cause of the leg cramps* that come on during and after the celebrationary dinner, for instance in athletes at a 'show' during the evening after the contest. It is especially liable to come on during tennis where the custom is for a player to drink between sets and games. We have all read in the daily papers of the cramp that periodically affects tennis players—there have been some instances in the last two years. Marathon runners are well aware of it and its remedy. They, especially, are liable to cramp on drinking water, hence the 'Marathon drink' (orange juice with glucose and water and ½ teaspoonful of salt). This is the drink that should be used after games and between sets. The cramps are, of course, identical with those of miners and stokers.

Vomiting may occur and then a vicious circle is set up.



Then mental confusions and obvious dehydration become apparent. Because of glucose lack, the body metabolises fat faster than it can utilise it, and acetone bodies form and eventually the alkalosis, with concomitant rise of blood urea and *all* signs of nephritis, seems to cancel out against the acidosis—at any rate some sort of equilibrium is reached and recovery follows if the picture is not complicated by heat effects. The above is a slight over-simplification of the known events for the sake of brevity to enable this paper to cover a wider field of syndromes than would be possible if too much detail were discussed. It is worth noting that the vomiting may go on for days—I have known one case vomit at short intervals for five days. Increasing blood viscosity and circulatory collapse may occur but has not previously been reported in athletics. The treatment is, of course, administration of intravenous NaCl and glucose, and the prevention the taking of the 'Marathon drink' in liberal quantities.

6. *Heat Effects.* Most common in Marathon runners. These are the same as are met with, much more frequently, in industry.

1. *Heat stroke* (rare) due to a failure of sweating and progressive rise in temperature, increased metabolism, more heat and greater failure of the heat regulating mechanism. Death may occur in hyperpyrexia, or permanent damage to the central nervous system (damage to the intelligence, or paralyses) may result. The skin is dry and hot and rectal temperature high.

Treatment. Emergency douching with cool water and turning fans on the sufferer. Cerebral malaria has to be excluded when making the differential diagnosis.

2. *Heat exhaustion.* Not rare in athletics. Stupor, shallow breathing, hypotension and postural fainting occur. How much of this is due directly to heat and how much to water and salt deprivation is not known, but the latter must play a very great part.

Treatment. Intravenous administration of normal saline may be needed.

Predisposing factors are, lack of acclimatisation, lack of fitness, and alcohol and insufficient sleep.

7. *Valsalva Experiment.* In weight lifters one may rarely meet with a case of sudden loss of consciousness due to this manoeuvre. If severe effort is made with a closed glottis, as in lifting a heavy weight, the intrathoracic pressure rises so much that the return of blood via the great veins leading into the thorax is impeded. Less blood reaches the heart, the cardiac output falls, the pulse becomes small and may disappear, cerebral

* Doubtless muscle strains and soreness and inadequate clearing of breakdown products of muscle action also play a part.

ischaemia and loss of consciousness ensues. The loss of consciousness may be very sudden and of course a fall results, with the possibility of injury from the weight or contact with the floor.

The differential diagnosis is from carotid sinus sensitivity, the usual tests for which have to be performed but will not here be discussed.

8. *Psychogenic Effects.* I think it is fair to state that many athletes who excel at the 'rapid' sports such as sprinting, are cast in a 'nervous' mould and, unless well balanced psychologically, are prone to neuroses, major and minor, hysterias, fugues, neurasthenic syndromes, da Costa's and effort syndromes, etc., and I only mention these because one is, as a doctor, often called upon to deal with such, and one must be aware of them, differentiate them from dehydration syndromes, etc., and from genuine cardiac disorders.

9. *Fatigue.* Little need be said about this. I would like, in regard to its pathogenesis, which is little understood, only to quote from S. Wright. Diagnosis and treatment, etc., need no enlarging upon.

'In steady prolonged exercise, fatigue is due to a number of ill-understood factors; in the main it is attributed to changes in the brain, resulting from slight anoxia and increased H-ion concentration. Afferent impulses set up in the active muscles (in part perhaps by the local physico-chemical changes) give rise to discomfort and contribute to the sense of weariness. Some of the stiffness may be due to swelling of the muscle accumulation of exuded fluid from the blood (muscle volume may increase 20%)'.

10. *The Heart.* I come now to a most controversial subject —the effect of severe physical exertion on the heart. The literature is most unhelpful mainly because authors will not state whether or not they are talking about trained or untrained hearts, or even healthy or unhealthy hearts. There is no argument that the diseased heart may suffer. Two instances may here be quoted.

The one is the death of a noted rugby player a few years ago, an hour after a strenuous match. From Dr. E. Jokl I have the information that autopsy showed narrowing of the coronary arterial ostia. He had complained of tightness of the chest anteriorly during previous games, was noted as a very hard and vigorous player. He, then, was apparently in training, yet with a defective heart.

The other case is that of a man of 44, in the army, untrained (sedentary) who played a pick-up game of soccer and sustained a coronary thrombosis next morning. This is a comparatively common type of occurrence however. Effort precipitates capillary rupture and haemorrhage in the atheromatous plaque, and hours or days later, thrombosis over the plaque ensues.

Apart from these the literature is full of 'Heart Strain', 'Soldiers' heart', 'Acute dilatation of the heart', 'Hypertrophy of the heart due to exertion', etc., etc. I cannot winnow it all here for you, but will endeavour to summarise the position and, for the sake of brevity, give my own opinions to the neglect to some extent of those of others. I will begin, however, by quoting Frederick Price, as his is the antithesis of my own views:

. . . In my opinion, cardiac strain may be the direct result either of temporary or long continued excessive physical exertion.

Symptoms. When an individual engages in temporary excessive physical exertion, there is stretching of the muscular fibres and dilatation of the cavities, together with an increased output of blood. Under normal conditions the dilatation is temporary and physiological. In some cases, however, the dilatation may persist, i.e., there is pathological acute dilatation. There may be lividity or cyanosis of the face, giddiness, and vomiting later, there may be collapse, quickened shallow breathing, coldness of the skin, precordial uneasiness or actual pain, and a quickened, small and thready pulse, while later, there may be evidence of pronounced cardiac dilatation, involving both sides of the heart. With rest, the cavities gradually return to the normal, but functional disability may continue for some time and in some cases the individual is never able to engage in physical exertion previously possible without experiencing some abnormal subjective symptoms.

In the case of strain, the result of long continued excessive physical exertion, there may be manifestations of cardiac hypertrophy, secondary dilatation, the usual symptoms of cardiac failure, and it may be a mitral or tricuspid systolic murmur, atheroma, and primary chronic or sclerotic endocarditis. Reduplication by the second sound, it may be only after exertion or on lying down after such, is sometimes present.

Prognosis.—When due to temporary excessive physical exertion, in the young and robust and the less severe cases, full recovery is the rule; this may occur in a few days or weeks, but on the other hand progress may be slow. Occasionally, however, as stated, the individual is never able to engage in physical exertion previously possible, without experiencing some abnormal subjective symptoms. In the more severe cases, the latter is not infrequent.

When the result of long continued excessive physical exertion, progress may be very slow, and full recovery is infrequent, and perhaps only occasional, while in the more severe, even the duration of life may be shortened.

From his 'Etiology of Cardiac Enlargement' comes this quotation too:

Simple hypertrophy is a physiological reaction to continued overwork. Among the causes are aortic stenosis, essential hypertension, chronic fibrosis of the myocardium, some of the varieties of Bright's disease, and diffuse hyperplastic sclerosis. It may also be due to long continued severe physical effort, as in heavy manual labour, athletic training and military training, chronic adhesive pericarditis, especially if there are extra-pericardial adhesions, and possibly continued excitation of the cardia action, as in chronic thyrotoxicosis. In the case of the first, the hypertrophy is left sided; in the second and fourth it is at least chiefly left sided, and in the third it is likely to be general.

Actual cases and X-ray findings are not given.

I have examined a great many athletes and their hearts, and cannot confirm his findings.

Paul White says 'Physical work and exercise do not primarily lead to heart disease or heart damage except in the rarest cases, though they may precipitate and aggravate symptoms and signs of heart disease already present, and may temporarily exhaust the cardiovascular reserve even in a healthy individual, "Athlete's heart", etc., are usually effort syndromes and chloride lacks, etc.' Even he fails to say if his 'healthy' individual is or is not trained.

Lewis says unequivocally that sudden strain on the sedentary heart is harmful, that no one under the age of 18 should be submitted to severe training—both statements one can hardly refute, but I would have liked to see examples—nowhere can I find a description of the actual cases that have led him to these conclusions. In regard to weight lifters he is more specific—a pulse rate over 100 at rest, reduplication of heart sounds and pounding in the head on lying or stooping, he considers 'danger signals'. The reduplication is of the first sound and is, he says, due to dilatation of the right ven-

tricle and asynchronous action with the left. One is still left quite unsatisfied, however, at the lack of X-rays, of follow up, of assessment of antecedent heart disease, etc., and electrocardiographs. In short, with most authors there are many dogmatic opinions and few closely studied cases. Personally I have never seen a fit well-trained heart come to harm, nor a youthful sedentary or untrained heart. Some of these I have studied fully with X-rays and E.C.G.s, etc. The adult sedentary heart may perhaps come to harm, but there are few cases and facts to hand. I myself, three years ago played hard squash, having had no exercise for months, and during the game had multiple and uncomfortable extra systoles, indicative, I should think, of myocardial anoxaemia. Before and afterwards no ill-effects were disclosed. This happened a second time a few months later. It is a question whether coronary spasm and angina or coronary occlusion can be caused in such a healthy untrained heart—I cannot answer it for post-mortem findings are not available.

A pathological degree of hypertrophy amongst long distance runners has not been noted in Roessler's most exhaustive X-ray studies. Any increase in heart size has been physiological in degree and has disappeared within six weeks of cessation of training. As a matter of interest, Jokl and Meaker investigated the causes of death during, or soon after exertion on sports fields, and list the order of frequency of causes of death as follows:

1. Coronary arterial disease.
2. Acute coronary occlusion.
3. Degenerate disease of heart muscle (one would have liked more detail here).
4. Ruptured aneurysm of aorta.
5. Chronic inflammatory disease of the myocardium.
6. Ruptured aneurysm of cerebral arteries.
7. Ruptured heart. (Not stated if healthy.)
8. Ruptured congenitally diseased aorta.
9. Development abnormalities of heart.
10. Development hypoplasia of entire arterial system (such people may previously have shown a strangely high standard of physical efficiency).

Sir Adolph Abrahams, brother of the sprinter, says 'the heart reserve is so great that no effort, however great, can produce any harmful effect'. Fitness is again not stated.

To my way of thinking, it is not only the heart reserve that is so great, but there are also several other protective mechanisms. The main one of these is the fact that ischaemia and cramp of the somatic musculature will precede similar changes in the heart. The greatest and most sustained high speed effort of all is in the 440 yards race, and the stage is reached in this where a competitor just cannot make his legs move any more and he will halt or fall, long ere the heart suffers. The main way in which a heart may be expected to be strained is by the huge venous return to the right side of the heart. The pericardium prevents undue dilatation of the right side of the heart. Cerebral ischaemia may come to the heart's aid. Acute loss of consciousness has been known to occur and then of course the heart is subjected to no more effort. It is said of one famous runner (I have the facts from an eye-witness, his trainer) that in a 440 yards race, he was jostled on to the grass verge at 150 yards, paused, resumed the race 15 yards behind the others, sprinted all the way to win the race, and dropped unconscious as he touched the tape. He was none the worse. Cerebral anoxaemia or carotid sinus mechanism may have been the cause of unconsciousness. The carotid sinus mechanism helps. If the blood pressure rises too high, this sinus reflexly slows the heart by vagal stimulation and the blood pressure falls. The result is that a healthy, trained heart exceedingly rarely, in my opinion, becomes damaged temporarily or permanently by any known form of sport or physical exertion.

ACKNOWLEDGMENT

Permission to publish this paper from the Johannesburg Clinical and Pathological Club, before whom the paper was read in 1948.

VERENIGINGSNUUS : ASSOCIATION NEWS

CLINICAL MEETING OF THE SUB-GROUP OF THE ASSOCIATION OF SURGEONS, JOHANNESBURG, HELD ON 2 MAY 1950

Mr. Fleming presented a paper on double plating of long bone fractures. He pointed out that this was an ambulatory method of treating fractures of the diaphysis of long bones.

The bones which lent themselves to this method were the femur, tibia, humerus, radius and ulna. The method had been used on some cases in America and the method with a different technique was used by Wenger in New York. The principle of the treatment was the fixation of the fragments by bolting two internal splints across the fragments, thereby producing a join which was strong enough to take all the stresses and strains that an unbroken bone would be called upon to withstand.

Thereafter no attention was paid to the bone and as soon as the soft tissues were healed, the patient was allowed full function of his limb.

In the case of a lower limb he was allowed to walk 10 days after the operation; in the case of the upper limb, after a shorter period.

The value of the full restoration of function was stressed and it was shown that when function was fully restored, not only did the muscles not waste, but the bone actually united more rapidly. There was total absence of pain after the pain of the operation had subsided. There was rapid restoration of full movements of the joint.

This was illustrated particularly in the case of the knee joint, in fractures of the femur when the average patient had 90° of flexion some four weeks after his operation and full flexion three to four months after his accident.

Four case histories were given and a patient with a double plating of his radius was shown to the meeting. A table of results was shown including 10 femurs, two forearms, one humerus and two tibiae. The disability periods averaged 47.3 days for the femurs, 7-10 days for the fore-arms, 9 days for the humerus and 30 days for the tibia. These times compared favourably with the times given by the Inter-Departmental Committee on the Rehabilitation of Persons Injured by Accident, viz. femurs 135 days, forearms 63 days, tibia 176 days and humerus 79 days.

The special instruments manufactured for the carrying out of this operation were demonstrated and a film was shown indicating the technique.

The plates were inserted through a standard incision for the approach to the bone, but this incision was slightly longer than that used for single plating. Special curved instruments were used for applying the nuts on the far side of the bone.

In an interesting discussion which followed, *Mr. Edelstein* traced the endeavours of surgeons to find some form of fixation of fractures so that function could be restored at an early stage. He stressed that the chief complication of the treatment of fractured femurs was the stiffness of the knee, which was often a permanent disablement. He compared the other method in vogue of treating fractured femurs by means of the Kuntscher nail, and said that that applied particularly to the femur and its final results would still have to be assessed. He pointed out that much more work would have to be done on double plating before it could be recognized as an acceptable method of treating fractured bones, but that should it prove its early promise, it was likely to become a generally accepted method of treatment.

Mr. Hamman stated that he had been trained by a conservative orthopaedic surgeon and was averse to opening a fracture if it could possibly be treated by conservative means. He admitted, however, that there were some fractures which had to be plated, and had noted that callus formed on the sides of the bone away from the plate, as though the plate itself inhibited the formation of callus. He thought that the amount of periosteal stripping associated with double plating might be an inhibiting factor to the production of callus. He indicated that he would like to see more of this treatment and if its future results justified it, he thought that the method might well replace older and more conservative methods of treatment.

Mr. Moller also congratulated *Mr. Fleming* on the ingenuity which he had expended in designing the instruments and developing the technique of this method as well as the skill he had exhibited in the application of these double plates. He thought that such a procedure could never be generally adopted, but should be reserved for those who have the necessary skill and team set-up to carry out the rather complicated procedures. He stated that we must be careful not to transgress the laws of the principles of surgery as those laws were well tried and proven over a long period. The principles in a fracture of fixing the joint above and below the fracture was a sound one, and one must be fully convinced that any departure from this method was well proven before it was generally adopted.

Mr. Colsen mentioned that he had assisted Mr. Fleming with some of these cases. He thought one had to be not only a good carpenter, but have a considerable knowledge of the strength of the materials and a knowledge of chemistry. While appreciating that the results in the case of the femur were excellent, he felt that such a procedure was not justifiable in a foreign fracture and favoured a small plate in those cases which had to be operated on, as a more extensive exposure had to be made for a double plate.

In reply *Mr. Fleming* stated that in his opinion the extra exposure was fully justified in view of the decreased disability period and the rapid restoration of joint movements, especially in cases of fractured femurs. He also stated that the work was being done under a grant from the C.S.I.R. and that research was continuing in the direction of the experimental determination of how much periosteum could be safely stripped and the best materials to use for the plates and bolts.

MINUTES OF THE GENERAL MEETING (QUEENSTOWN DIVISION) HELD IN THE FRONTIER HOSPITAL ON 15 JUNE 1950

Present. Drs. Bosman, Holmes, Kaplan, Louw, Nel, Papilsky, Rifkin, Schaffer, Sapirstein, Schweitzer, Swemmer, Voortman.

Apologies of Absence. Drs. Simonsz, Weideman.

Chairman. Dr. Schaffer.

Finance. At the request of Dr. Louw it was agreed that finance be discussed first. The Secretary gave the financial statement which showed that the dinner had been run at a loss Members agreed to liquidate the deficit.

Health Services. Dr. Schaffer intimated that Federal Council had formed a Sub-Committee for Health Service in the Union. He read portions of the report which had been published in the *Journal*.

Dr. Louw thought that we should proceed more actively with the Medical Service and leave the Social Services; that the idea of free choice of doctor was overdone; that few persons cared. What they wanted was adequate and thorough medical attention. After further discussion the following resolution was passed *nem. con.*

'In the opinion of this Division it is not advisable for the Association to commit itself to any definite scheme for improved Health Services for the Union at present.'

Dr. Louw felt that we should be prepared to fall into line with the Government, but we should state our financial views, which were wanted. Hitherto we always carefully refrained from touching on the question of finance. Consequently the following resolution was put to the vote:

'In the opinion of this Division the Association should be prepared to co-operate in any satisfactory Health Scheme which will make it possible for every qualified medical practitioner to participate in such a scheme, should he wish to do so, at a remuneration commensurate with his state, or subject to the practitioner being compensated.' Voting: 6 to 5 *against*.

Medical Aid Society. It was noted that from 1 July 1950 for a period of one year there will be an overall reduction of the tariff by 10%. Dr. Schaffer mentioned a new development, viz. 'B Tariff' which will provide for the lower income group. It had been sponsored by the Natal Chamber of Industries, and included Indians and Coloureds.

CLINICAL CASES

1. Dr. Kaplan showed the skiagrams of a male European aged 39 years who had attacks of vomiting and dull pain in the epigastrium. Further observation was recommended, the case to be seen again.

2. Dr. Sapirstein showed an interesting case of a female Native with a rapidly growing neoplasm of the face and which had commenced in the palate. The nature of the growth was obscure. Dissection of one of the enlarged cervical glands for biopsy was advised; report at the next meeting.

3. A Native child age 7 years convalescing from typhoid; skiagram shows a large mass on the right side. A possible perinephric abscess.

4. A case of a male European aged 60 years, stone-mason by trade. X-ray picture of the chest showed pulmonary tuberculosis superimposed on silicosis.

The Chairman thanked those who had exhibited the interesting cases; the Superintendent of the hospital for the use of the Board room and the Matron for the excellent tea.

The meeting terminated at 10.30 p.m.

BARAGWANATH MEDICAL SOCIETY: PROCEEDINGS OF A MEETING HELD AT BARAGWANATH HOSPITAL ON 27 JUNE 1950

Mr. S. Kay demonstrated a case of Albright's disease. The features were polyostotic fibrous dysplasia affecting most of the bones of the skeleton, an area of pigmentation of the left buttock and thigh, precocious sexual development and bone age. The blood calcium and phosphorus were normal and the alkaline phosphatase was markedly raised.

Prof. J. Gillman said that this may be one of the diseases in which there is a strong familial tendency. He was interested in the fact that one of the patient's brothers suffered from deafness.

Fatal Hypoglycaemia in an Adult Bantu Male. The history, clinical findings, biochemical changes, the electrocardiogram and X-ray films were presented. The salient features of the case were progressive weakness, cough and peripheral oedema. The serum proteins were albumin, 2 gm., globulin, 3 gm.; the alkaline phosphatase, 34 units. The electrocardiogram showed no characteristic changes but the QT segment was 0.42 second.

Dr. M. M. Suzman discussed the possibilities of hyperinsulism due to hyperplasia, adenoma and carcinoma of the

cells of the islets of Langerhans. In addition he commented on the various causes of hypoglycaemia due to pituitary disease, Addison's disease and von Gierke's disease. He also discussed the important role the alpha cells of the islets of Langerhans played in blood sugar regulation. The liver played a very important part in carbohydrate metabolism and he suggested the cause of hypoglycaemia in this case be due to severe liver disease.

The autopsy findings (*Dr. D. P. G. Stewart*) were mediastinal and retroperitoneal tuberculous adenitis and tuberculous pericarditis. There was no evidence of carcinoma or adenoma of the pancreas. The liver weighed 1,300 gm. and the thyroid, pituitary and adrenals were normal.

Histology (*Dr. J. Higginson*) showed no evidence of carcinoma, adenoma or hyperplasia of the islet cells and there was no suggestion of an increase in the size or number of the islets.

The only change in these cells was by way of atrophy. The liver showed slight fatty change, siderosis and minimal fibrosis. The glandular and pericardial lesions were confirmed as tuberculous. The adrenals, pituitary and thyroid glands were normal.

Dr. A. D. Gillanders discussed the effects of malnutrition and the importance it played in heart failure. He also discussed further findings in his investigations of cases of liver disease and cardiac failure, laying special emphasis on the blood sugar curves recorded after stabilization on a standard hospital diet. These curves showed a low fasting level, impaired carbohydrate tolerance after ingestion of glucose and a severe degree of insulin resistance. Liver biopsies revealed the presence of a normal or excessive amount of glycogen yet the glycogen was not mobilized by adrenaline. The changes in the livers of the patients were one or more of the following:—fatty change, haemosiderosis and cirrhosis. He commented on the unusual combination of fasting hypoglycaemia and an adequate glycogen storage in the liver. Such a combination occurred only in von Gierke's disease but he stressed the differences between these two in other respects. *Dr. Gillanders* emphasized the role of the liver in carbohydrate metabolism and evoked a broader concept than its dominance in one organ. He suggested that the above abnormalities in carbohydrate metabolism might be based on malnutrition and that many cases of coma admitted to hospital were due to hypoglycaemia.

Dr. T. Gillman emphasized that carbohydrate disorder is a manifestation of disease in many organs and attention should not be directed solely to a single organ. He discussed the important role that connective tissue played in the disorder and probably a defect in this tissue existed in malnutrition.

Professor J. Gillman spoke on the wide variations in normality in the Bantu who, with a low protein and fat diet, have a carbohydrate mechanism set at a different level. The Bantu has a background of malnutrition and many abnormal conditions are already determined *in utero*. He stressed the importance of pursuing investigations to determine the wide range of apparent normality in these people.

JOINT MEETING, BRITISH MEDICAL ASSOCIATION AND MEDICAL ASSOCIATION OF SOUTH AFRICA, 1951

Scientific Papers

With regard to the announcement concerning scientific papers which was published in the first Congress Circular in June last, the Organizing Committee now advises that the policy has been revised to conform with that of the B.M.A., namely, the office-bearers of the various sections will draw up their programmes and invite speakers to present papers on the subjects chosen.

Papers on original work may still be sent in to the secretaries concerned but the final decision as to which will be accepted must rest with the Organizing Committee, depending on the limited time available. All communications should be addressed to the Secretary of the respective Section, c/o Congress Office, Medical House, 5 Esselen Street, Johannesburg.

H. Moross,
General Secretary,
Organizing Committee.

15 August 1950.

PASSING EVENTS

Tariff of Fees for Approved Medical Aid Societies

A new Tariff Book of Fees for Approved Medical Aid Societies has been drawn up incorporating the 10% reduction recently agreed to by the Association after consultation by questionnaire with its members. This is effective from 1 July 1950.

Copies have been posted to members of the Association, but extra copies may be obtained on application to the Medical Secretary, P.O. Box 643, Cape Town. Orders should be accompanied by a remittance of 9d.

* * * *

Dr. Edward E. Wood of African Life Buildings, St. George's Street, Cape Town, has retired from practice owing to continued ill health.

* * * *

Dr. K. Cunningham has returned from London where he attended the International Congress of Ophthalmology as a delegate for the South African National Council for the Blind.

* * * *

Mr. O. Popper, Senior Surgeon, Johannesburg Hospital, and of the Department of Surgery, University of the Witwatersrand, has been invited to teach at the International Course in Audiology to be held in Stockholm on 10 September.

Mr. Popper will be demonstrating the transtympanic approach in association with Professor Robert Lund of Copenhagen, who is delivering the *ex cathedra* lectures on the Popper operation.

Dr. Evelyn Popper, M.D. (London), M.R.C.P. (London), and Miss Janet Popper will be accompanying him and they intend to visit Rome, Vienna and Paris.

* * * *

Mr. William G. Schulze, formerly of Groote Kerk Buildings, Parliament Street, Cape Town, wishes to inform his colleagues of the following change of address from 1 September 1950:—

Consulting Rooms: Southern Life Buildings, St. George's Street, Cape Town. Telephone: 3-1322.

Residence: Hamilton House, St. James. Telephone: 8-5433.

Augmented Executive Committee in the Transvaal

Members of the Association in the Transvaal should note that the Augmented Executive Committee meets frequently. The Administrator of the Transvaal has appointed a permanent Committee consisting of the Medical Director of Hospitals, the Secretary of Hospitals and an official of the Hospitals Department, for the express purpose of discussing with the Augmented Executive Committee any problems which may arise in connexion with the Transvaal Provincial Hospitals. Members of the Association should, therefore, communicate with Dr. Braun, Chairman of the Augmented Executive Committee, Medical House, 5 Esselen Street, Johannesburg, when any problems arise in connection with their hospital work.

OFFICIAL ANNOUNCEMENTS

MEDICAL ASSOCIATION OF SOUTH AFRICA

Annual General Meeting

Notice is hereby given that the Annual General Meeting of the Medical Association of South Africa will be held at Medical House, 5 Esselen Street, Johannesburg, on Thursday, 12 October 1950, at 9.30 a.m.

AGENDA

I. Minutes.
II. Annual Report and Balance Sheet.
III. Election of Auditors.
IV. Other business.

A. H. Tonkin,
Medical Secretary.

Cape Town.
31 August 1950.

MEDICAL ASSOCIATION OF SOUTH AFRICA

FEDERAL COUNCIL

Notice is hereby given that a meeting of the Federal Council will be held at Medical House, 5 Esselen Street, Johannesburg, on 12 October 1950, at 10 a.m.

AGENDA

1. Notice convening the meeting.
2. Proxies.
3. Minutes of previous meeting (circulated).
4. Matters arising out of the minutes.
5. Financial statement by the Honorary Treasurer.
6. Report of the Executive Committee.
7. Reports of other Committees.
8. Reports deferred from the previous meeting.
9. Notices of motion transferred from the previous meeting.
10. New notices of motion.
11. Other business.

A. H. Tonkin,
Medical Secretary.

Medical House,
35 Wale Street,
Cape Town.
31 August 1950.

AMPTELIKE AANKONDIGINGS

MEDIESE VERENIGING VAN SUID-AFRIKA

ALGEMENE JAARVERGADERING

Kennis geskied hiermee dat die Algemene Jaarvergadering van die Mediese Vereniging van Suid-Afrika gehou sal word te Mediese Huis, Esselenstraat 5, Johannesburg, op Donderdag, 12 Oktober 1950, om 9.30 vm.

AGENDA

I. Notule.
II. Jaarverslae.
III. Verkiesing van Ouditeure.
IV. Ander besigheid.

A. H. Tonkin,
Mediese Sekretaris.

Kaapstad.
31 Augustus 1950.

MEDIESE VERENIGING VAN SUID-AFRIKA

FEDERALE RAAD

Kennis geskied hiermee dat 'n vergadering van die Federale Raad gehou sal word op 12 Oktober 1950, om 10 vm. in Mediese Huis, Esselenstraat 5, Johannesburg.

AGENDA

1. Kennisgewing van vergadering.
2. Volmagte.
3. Notule van vorige vergadering (reeds uitgestuur).
4. Sake vermeld in die notule.
5. Geldelike verslag deur die Ere-Tesourier.
6. Verslag van die Uitvoerende Komitee.
7. Verslae van ander Komitees.
8. Verslae uitgestel van die vorige vergadering.
9. Kennisgewings van voorstelle oorgedra van die vorige vergadering.
10. Nuwe kennisgewings van voorstelle.
11. Ander besigheid.

A. H. Tonkin,
Mediese Sekretaris.

Mediese Huis,
Waalstraat 35,
Kaapstad.
31 Augustus 1950.

REVIEWS OF BOOKS

BIOGRAPHY OF BACTERIA

The Cytology and Life-History of Bacteria. By K. A. Bisset, Ph.D. (Pp. 136 + xii. With 43 Illustrations. 18s. 6d. 1950.) Edinburgh: E. & S. Livingstone Limited.

Contents: 1. Introduction. 2. Technique and Cytochemistry. 3. Surface Structures. 4. The Bacterial Nucleus. 5. Reproduction. 6. Sexuality in Bacteria. 7. Life-Cycles in Bacteria. 8. Macroformation. 9. Bacterial Morphology and Classification. 10. The Genetics of Bacteria.

Most of us, when thinking of bacterial morphology, think in terms of appearances in films stained by Gram's method. Thus Streptococcus pyogenes is a grampositive coccus occurring in chains, and B. coli is a gramnegative bacillus. Bacterial morphology as revealed in preparations in which shrinkage during fixation has been minimized and special techniques have been adopted to stain cell walls and nuclear material is strikingly different. Bisset has made an intensive study of morphological problems. His book is an adequate summary of present knowledge and includes the results of many of his own and of other investigations previously published elsewhere.

The author rightly stresses the importance of a knowledge of the special staining methods and the structures they reveal. Of particular interest are the nuclear changes which can be observed during multiplication, the formation of resting cells, the formation of various stages of the bacterial life cycle and the visual evidence in favour of sexual fusion of nuclear material.

The book is a timely and welcome one in view of the growing interest in the study of nucleo-proteins of bacteria and viruses. Unfortunately, as is almost inevitable with an exhaustive study of a relatively limited subject, there is perhaps a tendency to attach too much significance to the special techniques employed. Thus the author suggests that electron microscopy is likely to be of very limited use in bacteriology. It is perhaps also a pity that the author enters the controversial field of bacterial classification—suggesting a redistribution of known bacteria into orders and families on the basis of his morphological findings rather than of our older concepts of bacterial structure.

It is nevertheless a book of real value to every bacteriologist.

COMMON SKIN DISEASES

The Common Diseases of the Skin. By R. Cranston Low, M.D., F.R.C.P.E., F.R.S.E., and G. A. Grant Peterkin, M.B.E., M.B., F.R.C.P. (Ed.). (Pp. 282 + x. With 148 illustrations of which nine are in colour. 4th edition. 21s.) Edinburgh: Oliver and Boyd.

Contents: 1. Introductory. 2. Diseases due to Animal Parasites. 3. Infections due to Pyogenic Organisms. 4. Fungus Infections. 5. Urticaria and Allied Conditions. 6. The Erythemata and Allied Conditions. 7. Hydroa. Vesicular and Bullous Eruptions. 8. Seborrhoea and Allied Conditions. 9. Alopecia Areata. 10. Dermatitis, Eczema and Allied Conditions. 11. Scaly Eruptions. 12. Scaly Eruptions. 13. Tuberculosis of the Skin. 14. Naevi. 15. Benign Epithelial Neoplasms. 16. Malignant Epithelial Neuplasms. 17. Benign Connective Tissue Neoplasms. 18. Malignant Connective Tissue Neoplasms.

This book fulfills its purpose admirably. The rarer dermatological conditions are omitted, but those which are more commonly encountered and may prove so puzzling and intractable in the hands of the unenlightened, are dealt with clearly, concisely and adequately.

In the chapter on fungus infections, *Tinea pedis* is not referred to as such, but is included in the section on *Tinea cruris*. It well merits a section of its own and mention might also then be made of the fungicidal fatty acids, so widely used to-day in its treatment.

There is a section devoted to eruptions due to the more commonly used drugs, and here no mention is made of the fixed drug eruption due to phenolphthalein. In view of the popularity of this aperient in various laxative tablets, it would be as well to include this drug and describe the characteristic features of the eruption it may produce.

Dermatitis, eczema and allied conditions are dealt with in a logical manner and the division of dermatitis into those of external and those of internal origin assists in clarifying a

subject confusing to so many. Infantile eczema is discussed in some detail and the treatment of this distressing condition is outlined lucidly and logically.

No distinction is drawn between 'leucoderma' and 'vitiligo' but it would perhaps be better to regard 'leucoderma' as any area of white skin occurring secondary to inflammatory lesions such as the leucodermic macules of syphilis or similar macules that occasionally mark the resolved lesions of psoriasis, lichen planus, pityriasis rosea and so on, whereas 'vitiligo' be applied to the idiopathic variety of primary leucoderma.

In the section on verrucae, the use of the cautery and curette might with advantage be included in describing the treatment of the common and the plantar wart.

The work is profusely illustrated, with large, clear and well-defined white and black, as well as coloured, pictures of almost every condition described; the print is large, on glossy, thick paper, so that the whole makes for pleasant and easy reading.

It is a book that can be strongly recommended to those for whom it has been written.

Otolaryngological History

A History of Oto-Laryngology. By R. Scott Stevenson, M.D., F.R.C.S. (Ed.), and Douglas Guthrie, M.D., F.R.C.S. (Pp. 155 + vii. With 52 illustrations. 17s. 6d.) Edinburgh: E. & S. Livingstone Ltd. 1949.

Contents: 1. Introduction. 2. Ancient History. 3. The Middle Ages and the Renaissance. 4. The Seventeenth and Eighteenth Centuries. 5. Otology becomes a Science. 6. The Education of the Deaf. 7. Laryngology in the Nineteenth Century. 8. Modern History. Bibliography. Index.

This interesting book represents an extraordinary amount of work and research into historical documents in many languages. It is well illustrated with many reproductions of portraits of men whose names have become bywords wherever otorhinolaryngology is practised.

Many of the things which we accept to-day in ear, nose and throat practice, and look upon as fairly modern discoveries, were known to medical men centuries ago. In ancient Hindu surgery, new noses were fashioned by flaps from the cheek or forehead to replace those cut off as a punishment for adultery. The drug ephedrine was known in China many hundreds of years before it was used in either Europe or the U.S.A.

The evolution of the complete examination of the ears, nose and throat as we know it, makes interesting reading—the difficulties encountered and surmounted, and how a singing teacher named Manoel Garcia became the first man to see the inside of the larynx using a mirror.

Dealing with tonsillectomy, the authors state that the first description of this operation was by a Roman (Celsus) in 30 A.D., using his finger as a dissector. The guillotine, so extensively used in the past, and still popular in certain countries, appears to be gradually fading out of use—modern surgeons favouring dissection as a surer method of removing the tonsil completely.

It is only in recent times that the three branches comprising the specialty have been brought together; but with modern advances in thoracic surgery, new discoveries of the mechanism of the labyrinth, etc., one wonders if the branches may not again split up in the future.

The Asthmatic Child

The Asthmatic Child: The Prevention of Asthma by Simple Home Methods. By G. F. Walker, M.D., F.R.F.P.S.G., D.C.H., M.R.C.P. (Pp. 20 with 24 illustrations. 2s. 6d.) Bristol: John Wright & Sons Ltd. 1950.

Dr. Walker has written a little pamphlet which will be of assistance to the asthmatic child. The booklet is aimed at parents who have the problem of ensuring that an asthmatic child should help himself to the fullest extent by practising simple exercises in the home.

It should serve this purpose very satisfactorily.

CORRESPONDENCE

Medical Fees and the Cost of Living

To the Editor: I should like to bring to your notice the present most unfavourable conditions under which general practitioners are struggling to make a living as a result of the greatly increased cost of living and the low value of the £.

Let us compare the years 1939 and 1950. In the former year a motor-car (one of the chief necessities for a general practitioner) cost about £300. To-day it costs approximately £900. Formerly, a general practitioner could theoretically buy a motor-car after seeing 600 patients at 10s. 6d. each. To-day if he still charges 10s. 6d. per visit, he must see 1,800 patients in order to purchase a car! If he wants to buy a car after 600 consultations, he must to-day charge £1 10s. per consultation.

Another simple way to realize how low our present charges are is to consider the value of clothing in relation to our fees. In 1939 one visit was equal to the value of one shirt or four pairs of socks. To-day *three* visits are only equal to the value of one shirt or to about five pairs of socks. At the present time the fee earned by a visit will hardly buy a tie, whereas in 1939 it was equal in value to four ties. Therefore, to keep the value of our charges for visits equal to the value of clothing, we must charge three times as much as we did in 1939—again we arrive at the figure of £1 10s. per visit.

In every direction our expenses have been doubled or trebled. The £ is to-day in fact only worth 6s. This means that when we charge 10s. 6d. per consultation, we are in fact only earning 3s. 6d.

In the past the medical profession to a large extent carried the poor on its back. To-day we are carrying not only the poor, but the middle-income group and the rich and we shall continue to do so until our charges are raised to their proper value.

We will, of course, not raise our fees to £1 10s. per visit as we should really do, but surely it is about time that we did something about the present grossly inadequate and ridiculously low fees that we are charging.

'General Practitioner.'

27 July 1950.

Thyroid Treatment of Hypertension

To the Editor: I do not wish to prolong correspondence about Dr. Menof's paper on the treatment of hypertension but I am writing this letter because Dr. Menof, in his reply to my previous communication, seems to have missed the whole point of my remarks. I do not question the results of his treatment; I only ask: What condition or conditions has he been treating? The definition of hypertension given in his letter does nothing to clear up the answer to this question. I would also point out that his definition of essential hypertension as 'a psychosomatic disorder characterized at first by a "functional" rise in blood pressure due to vasoconstriction . . .' is a very different proposition from the criterion he gave in his paper on the treatment of the condition, in which he stated that he treated all cases of hypertension irrespective of what the cause of the elevated blood pressure might be and that no effort was made to discriminate.

This new definition makes the problem even more baffling to the reader. In any generalized condition of vasoconstriction the arterioles of the kidney will be affected with those of the rest of the body and sooner or later renal function will be affected and a hypertension with renal involvement will occur; yet in these cases Dr. Menof's treatment was not successful.

It is well known that most cases of essential hypertension are symptomless until cardiac, renal or encephalopathic symptoms occur (headache being regarded as a sign of encephalopathy—see Paul White's *Heart Disease*, p. 436).

Once the patient is aware that he has hypertension the picture is often clouded by psychosomatic symptoms and it is often very difficult to differentiate such cases from purely psychosomatic states such as da Costa's syndrome. Even in cases which are classified by Dr. Menof as being in the 'later stages—years after its onset' this difficulty persists and Dr.

Paul White (*Heart Disease*, p. 438) states that even when hypertension is present, a case cannot be termed hypertensive heart disease unless there is also present cardiac enlargement due mainly to left ventricular hypertrophy.

Dr. William Evans (*Cardiology*, 1948, p. 204) states: 'The following definition is meant to be a guide in the diagnosis of hypertension. The blood pressure is raised when the systolic pressure is 180 or over and/or the diastolic pressure is 110 or over on three consecutive examinations, and in the presence of clinical, radiological and cardiographic evidence of cardiovascular hypertrophy'.

Most of us will feel that Dr. Evans sets the standard too high as regards the diastolic pressure, but the fact that he does so is an indication of how difficult he has found it to be sure of the diagnosis if he uses a lower level of diastolic pressure as his guide. His insistence on the presence of signs of cardio-vascular hypertrophy may astonish Dr. Menof but, in view of this writer's wide clinical experience, it should merit his consideration.

If we are going to label disease syndromes with names which are used universally, it is essential that we only apply these names to conditions which fulfil the universally accepted diagnostic criteria of the disease. Unless we all speak the same language it is bound to lead to confusion about what pathological condition is being discussed.

For my part I have often found essential hypertension a very difficult condition to diagnose with certainty and I am sure there must be many like me who would hesitate to make such a diagnosis until we have had our patient in hospital and under observation for a period.

J. C. Gilroy.

Baragwanath Hospital,
Johannesburg.
28 July 1950.

ACUTE LARYNGO-TRACHEO-BRONCHITIS

To the Editor: Several cases of this relatively rare clinical entity have been reported in the *British Medical Journal* recently,[1, 2] the following may be of interest.

On 14 February of this year I was summoned to a case of laryngeal obstruction in a child aged 15 months. The medical practitioner who referred the case asked for it to be treated at home if at all possible as the baby was an only child and he did not consider it an emergency.

Within an hour of the baby first having been seen I examined it. Fairly marked stridor with rib and sternal recession was noted. The child was pale and listless with a thin, racing pulse, obviously a candidate for tracheotomy. The mother told me that the child had been off colour for 24 hours and that it was only after her house-doctor had left that the child's breathing had become so bad. The baby was admitted to hospital immediately and the theatre warned to prepare for tracheotomy.

On admission to the ward, the child's condition having improved somewhat, it was treated as only a threatening diphtheritic laryngeal obstruction, but at 4 a.m. the following morning an emergency tracheotomy had to be performed. On inserting the tube much pus welled out of the trachea.

Following the tracheotomy there was not the dramatic improvement in breathing that one usually sees in true laryngeal obstruction. There was still a certain degree of stridor and the inner tube had to be cleaned of pus every half-hour. On 15 February the child was very toxic and there was still rib recession with poor air entry to both lungs. All this time the baby had been on strenuous anti-diphtheritic treatment, receiving 200,000 units penicillin three-hourly in addition.

On 16 February there was slight improvement and there appeared to be less pus in the trachea but on the following day the child succumbed.

Three negative swabs for *C. diphtheriae* and one negative for *Strep. haemolyticus* were obtained. At autopsy no more than a confluent bronchopneumonia could be found.

Obstructive symptoms develop very rapidly in acute laryngo-tracheo-bronchitis as well as in laryngeal diphtheria, and it appears that if there is not a rapid and dramatic improvement following tracheotomy, one must treat for the former condition as well as the latter. Continuous suction through the tracheotomy tube or frequently repeated bronchoscopic suction is advised, with intensive chemotherapy using aureomycin as well as penicillin. No single organism but a very mixed bag is to be incriminated in this condition, death being due to the intense toxaemia as well as to the drowning in pus.

It is of great satisfaction to learn that a case may be saved by prompt intervention along these lines, as indicated by our British colleagues.

REFERENCES

1. Brit. Med. J., 11 March 1950, p. 578.
2. Brit. Med. J., 1 April 1950, p. 786.

T. H. Stewart,
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31 July 1950.

INTESTINAL CLAUDICATION

To the Editor: I must congratulate both Mr. Wilkie on his scientific article and the Editor on the high standard of reproduction achieved (this Journal, 1 July 1950, *Massive Resection of Small Gut*).

There seems to be one small error in Mr. Wilkie's deduction about the collateral circulation. He assumes (and probably correctly) that the old primary thrombosis of the superior mesenteric artery was compensated partly by reversed flow of blood along the marginal artery of Riolani so that blood could flow from the inferior mesenteric into the middle colic and thence into the main stem of the superior mesenteric artery. Note that this collateral flow of blood enters the superior mesenteric artery high up *proximally*, before it has given off its first arcades to the small gut. Yet Mr. Wilkie assumes that the large anastomotic artery which seemed to be nourishing the *distal* ileum came directly from this anastomosis. I cannot accept this. Actually I find it difficult to conceive the source of the large anastomotic artery to the terminal arcades. Normally the sole link-up of these arcades is with the ileo-colic, itself a branch of the superior mesenteric.

Mr. Wilkie has done well to draw attention to the two syndromes of vascular obstruction in gut, i.e. not merely the syndrome of complete vascular insufficiency or occlusion but also the syndrome of relative vascular insufficiency or claudication.

Such papers as those of Mr. Wilkie are helping to establish recognition of mesenteric vascular disease. It is strange that this recognition should have been so tardy. More than 150 years ago the elder Heberden described angina pectoris, the syndrome of coronary claudication. A century more was to pass before the syndrome of absolute coronary occlusion was noted. These same vascular syndromes apply to the brain, the limbs and to the gut. Now, at last, claudication of the gut is being recognized, though the conception of absolute mesenteric occlusion has been accorded lip service, at least, for 50 years.

In Chemistry Mendeléeff conceived the brilliant and subtle hypothesis of the Table of Elements. Based on this hypothesis he told chemists to go out and look for the missing elements to accord with his theory; and now nearly all these elements have been found. Here is a simple pathological theory whose truth seems self-evident: Tissues whose blood supply is absolutely impaired will have their function absolutely impaired and tissues whose blood supply is relatively impaired may have their function relatively impaired. Is there no minor Mendeléeff among us who could have listed all important organs and prophesied lesions of them on the basis of this simple theory? This should have been done long ago; and clinicians could have gone out to find the missing diseases. They would not have had to look far.

From a practical standpoint it may be asked how mesenteric vascular disease may be diagnosed. It is easy to diagnose in the presence of the causative heart failure or endocarditis; but it may be caused by blood dyscrasias, portal hypertension, mesenteric arteriosclerosis, and idiopathic or unknown causes.

I operated on a case where the whole territory of the superior mesenteric was occluded as a result of pressure within the leaves of the mesentery by tuberculous nodes. The following features are very suggestive of mesenteric occlusion when they occur in combination:—

1. Signs of obstruction.
2. Marked shock which fails to respond to massive blood transfusion.
3. Weak or absent peristaltic sounds.
4. Melaena.
5. High leucocytosis with a relative polymorph increase.
6. X-ray signs of ileus in the small gut but, contrary to the usual finding in mechanical obstruction, the large gut shows normal faecal and gas content. Here I must urge that all plain X-rays of suspected mesenteric occlusion should be taken before diagnostic enemas have been given a chance to wash out the large gut.

The diagnosis of relative mesenteric vascular insufficiency or claudication is even more difficult. Usually it is diagnosis by exclusion and, as in Mr. Wilkie's case, it is often a retrospective diagnosis. The following combination is suggestive:—

1. An ageing arteriosclerotic patient.
2. Recurrent attacks of subacute abdominal pain with obstruction.
3. Intervals between attacks marked by indigestion and constipation.
4. Immediate investigation shows nothing suggestive of another lesion such as carcinoma or diverticulitis.
5. The presence of carcinoma is still further excluded by the eventual long course of the disease.

Slowly, then, the diagnosis of intestinal claudication dawns in the mind of the practitioner. In fact it would be a pity if the diagnosis were to dawn too quickly; all other possibilities must first be exhausted, otherwise remediable lesions may suffer a tragic neglect.

Personally I am of the opinion that claudication of the gut may be a fairly common disease. It is an opinion that would be difficult to prove; for social reasons most of these cases run their course under the eye of the general practitioner. Few are ever submitted to the more rigid diagnostic criteria of clinical research centres. At present the diagnosis of mesenteric claudication is rarely made. Perhaps it is better so; when the pendulum swings to the opposite extreme it may become dangerously overdiagnosed.

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27 Terminus Street,
East London.
5 August 1950.

STILL NOT FUTILE RADIOLOGICAL PROCEDURES

To the Editor: Having read Dr. V. Berman's reply (29 July) one wonders why he wrote his original letter (17 June).

In his answer (29 July) Dr. Berman takes exception to Dr. Hirschon's question: 'Does Dr. Berman also consider routine fluoroscopic examination of the chest preliminary to a barium meal, to be a "futile procedure"?' on the grounds that 'surely nobody could regard this procedure and the one at issue as analogous'.

Apparently they can and do! After all, Dr. Berman's qualification that he 'would never presume to say that no intrathoracic pathology existed merely because he had not detected anything on the screen' in no way minimizes his admission to the routine performance of an obviously imperfect, and possibly misleading, procedure.

Furthermore, Dr. Berman would, like other Cape Town radiologists, report that the chest on fluoroscopic survey appeared normal or clear, and he would just like other Cape Town radiologists, feel that he was not thereby 'lulling the clinician into a false sense of security by a report that (in this case) the chest was clear on screen examination' (his own words).

On 17 June Dr. Berman stated: 'It is very often difficult enough to detect subtle changes on a first-class radiograph' yet Dr. Berman takes exception when experienced radiologists look for gross changes on sinus screening, in the same way as they and Dr. Berman look for gross changes when screening the chest; for, as Dr. Berman admits, he (and presumably Cape Town radiologists) 'would never presume to say that no intrathoracic (or sinus) pathology existed merely because they had not detected anything on the screen' and, therefore, all that either could say is that the chest (or sinus) was clear on screening.

Dr. Berman's reply (*sic*) to *Clinician* stated: '*Clinician* has said nothing which merits serious consideration.' All *Clinician* said with regard to routine accessory sinus screening and Dr. Berman's insistence on 'excellent radiographic studies in several views' with which 'it is very often difficult enough to detect subtle changes' was that 'these limitations have never been considered symptomatic of the futility of either of the above procedures' nor, one should now add, of screening of the chest before a barium meal; nor, for that matter, of any of the numerous routine investigational procedures performed daily in the service of diagnosis.

It is quite correct that what *Clinician* said does not 'merit serious consideration.' The average, normal variety will suffice to recognize the validity of the above principle which is also inherent in Dr. Berman's own admission to his routine use of the imperfect, and possibly misleading, preliminary chest fluoroscopy in barium meal examination. *Q.E.D.*

Clinician.

Cape Town.
9 August 1950.

THE NEW MEDICAL AID SOCIETY TARIFF OF FEES

The Editor: I have received the new *Tariff of Fees for Medical Aid Societies* and have been not a little pained at the changes made in the section on *Minor Procedures*. In particular, I am wondering why the fee for the electrocardiogram has been reduced from 1½ guineas to 19s. In the first place, no distinction has been made between the fees of the G.P. and the Physician. Very few G.P.'s use the ECG, and it is essentially a machine to be used by a specialist. The ability to interpret the new unipolar chest and limb leads requires a high degree of skill and time, and I think that most doctors will agree that it is in the specialist's province that the ECG lies.

In the second place, whether most doctors will agree with the foregoing or not, is not so important. What is important is that the fee for the ECG is the same as that charged for a diagnostic paracentesis of the chest or abdomen. The latter procedures can be performed by any medical student after 15 minutes' instruction. It takes me 15 minutes to take an ECG record but the interpretation of the record may take another 15 minutes with much puzzling over changes not easily understood. I have to take three limb leads, six unipolar chest leads and three unipolar limb leads but I am not paid for the physical labour entailed in taking the record. This can be done quite satisfactorily by my receptionist now; the interpretation of the record is what I am paid for.

To me it appears that an ECG is worth the full 2 guineas which I charge my private patients. Whoever was responsible for this change in the tariff is not a physician, or does not practise electrocardiography. In any case he is ignorant of the skill and knowledge needed in the interpretation of the modern ECG.

Physician.

14 August 1950.

BACK NUMBERS OF THE 'LANCET' FOR 1949

To the Editor: To complete my set of the *Lancet* for 1949, I need the copies dated 1, 8, 15, 22 and 29 January and 5 and 12 February. I would be very grateful if any of your readers who have these copies but do not wish to retain them, would sell them to me. Would they please communicate with me at the address below.

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